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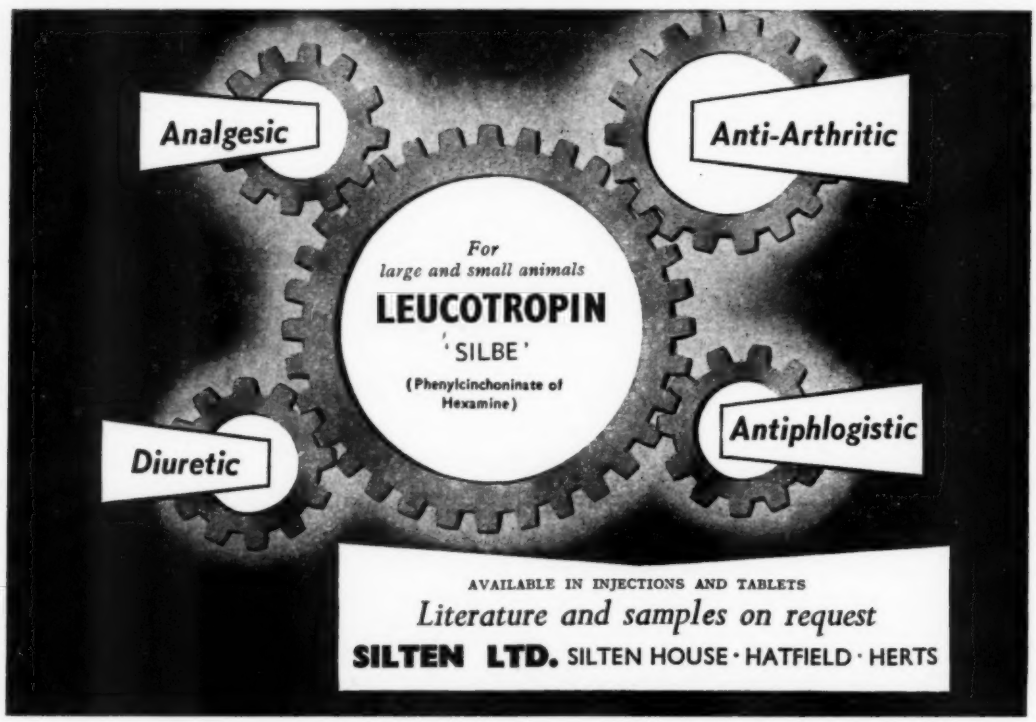

The
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The President's Address to the Association

ON Monday last, the opening day of the Annual Congress, the President delivered the traditional Address to the members of the Association, Mr. Callender, who was received with loud applause, said:—

"The rules of this Association would appear to require the President to deliver an address prior to handing over the chair of office. In any case it appears to have become such a firmly established custom, that however much one would wish to avoid it there seems to be no means of escape. It is, I can assure you, an ordeal; but on reflection it occurs to me that it might be somewhat of an ordeal for you to listen to me. If it should prove too great, please endeavour to bear with me in the knowledge that it is no wish of mine that I should inflict an address upon you at all; I am merely bowing to what has become an established custom in the conduct of our affairs.

This, I note, is a time when the President can express opinions and make predictions and statements without having to defend them in any subsequent debate which normally follows such utterances.

Before getting down to details, however, there are two things I particularly want to say. The first is, how gratifying it is to us all to be holding our Congress in this ancient University City of which the School of Veterinary Medicine, officially opened by the Queen in October, 1955, is its newest place of learning. The School has on its teaching staff a notable body of scholars and research workers, and I am quite certain that the students and graduates from it will be fully worthy of those who have taught them. Then, I would like to say from us all how very greatly we appreciate the presence at this Congress of a number of most notable scientists from outside the profession who have been good enough to give us the benefit of their learning in those plenary papers we are so much looking forward to. This is an innovation which I hope will be followed in other years.

The Present Position

It is customary, on an occasion such as this, to review the progress of the profession in general, and this Association in particular; even to endeavour to forecast what the future might hold for us. Such forecasts may vary considerably: sometimes optimistic—sometimes the reverse. At the risk of being branded as somewhat of an optimist, I am going to express the opinion that the prospects of our profession were never greater, provided that we are ready and willing to grasp the opportunities which occur; that we are ready at all times to assume those responsibilities which are rightly ours, and that we ensure that we are fully equipped to meet the many and varied problems with which as a profession we are continually called upon to deal.

That we do our best to keep our clinical and scientific knowledge up to date is, I think, pretty well evidenced by the variety of the contents of our official journal week after week. Additionally, we provide to members of the Association up-to-date handbooks prepared by the Technical Development Committee. Then there are sectional meetings, such as those of the Association of Veterinary Teachers and Research Workers, and the host of practical papers read at Divisional meetings.

Indeed, we endeavour to keep abreast with advances in other branches of science, and here I quote, 'that work is being done at present by our colleagues at Compton in investigating the uptake of radio-active strontium by certain crops, the ingestion of which might be injurious to grazing animals and from them to the human population.' Work is also in progress to determine the effect of mixed fission products administered to cattle. I cannot refer to this work in detail as it is still being actively pursued but a preliminary report will be presented at the International Conference on Radioisotopes in Scientific Research in Paris next month. I feel this work will provide fundamental knowledge for the guidance of farmers and others, should our land ever become contaminated seriously with the fall-out from atomic explosions.

Increased Demand for Services

There is undoubtedly, in recent years, a greatly increased demand for the services of the veterinary surgeon. That demand may be partly due to the

enhanced value of livestock, but it is mainly due, I think, to two factors:—

Firstly, the stock-owner has become very much more disease conscious, particularly in regard to the less spectacular diseases; and he is less prone to regard losses from such diseases as inevitable. Secondly, he has much greater confidence in the ability of his veterinary surgeon to help in solving the many disease problems which, if left unsolved, result in great financial loss. In this connexion it is of interest to note that whilst foot-and-mouth disease, one of the so-called spectacular diseases, costs the country on an average half a million a year, the losses from mastitis have been estimated at millions annually, and those from infertility are, according to some estimates, even greater. The emphasis is more and more on the prevention of disease, and the question posed many years ago ' if preventable why is it not prevented?' is just as applicable to-day as it was then.

The Animal Health Division of the Ministry of Agriculture, Fisheries and Food are concerned with prevention, control, or eradication of certain diseases which are the subject of legislation, or in respect of which schemes of control or eradication have been devised. In these schemes there seems little doubt that the practising veterinary surgeon must continue to play an important part. The Inter-Departmental Committee on Veterinary Education stated in its report in 1938 that ' the veterinary practitioner is the physician of the farm and the guarantor of the nation's food supply. His established title of veterinary surgeon obscures his more important functions which are not only to cure disease among farm stock, but also to check the spread of disease; to eradicate it when established, and by advice in breeding and nutrition to build up a stock as resistant as possible to disease.' These words, I submit, are just as true to-day as when they were written nearly 20 years ago; possibly they are even more applicable now.

Preventive Medicine

In the field of preventive medicine the veterinary profession has a record of which it can justly be proud, and in the term preventive medicine we must include those measures to control the epidemic diseases which still constitute such a menace on the continent of Europe and in many parts of the world. Let us reflect for a moment on the position in this country regarding these diseases, for the achievements in this connexion are often lost sight of. Cattle plague, pleuropneumonia, glanders, parasitic mange, sheep pox and rabies are now of academic interest only, apart from the odd case of rabies which has occurred over the years in imported dogs undergoing quarantine.

In this connexion, and as my words may be carried through the Press or the wireless to a larger public, I would like to emphasise the crucial importance of the 6 months quarantine. From time to time people whose sympathy for animals exceeds their practical knowledge make pleas for quarantine to be diminished or even done away with. Such a course would be extremely rash. Rabies may take a long time to develop, and cases have been known even after the full 6 months have been spent in quarantine kennels. Do let me, therefore, make clear that quarantine regulations are vital to the safety of the public.

To the list of diseases eradicated, sheep scab might be added, for apart from an odd outbreak in imported sheep within the confines of the various landing places no outbreak has been confirmed in this country since 1952. Credit is often given for this happy state of affairs to the advent of the BHC dips, which are undoubtedly of immense benefit to the sheep farmer; but I would submit that despite the advent of these dips it was the unremitting efforts of the veterinary staff of the Animal Health Division, which brought it under control, and in fact eradicated it before the new dips became fully available. It is still prevalent in other countries which have access to the new dips.

Another distinct achievement is the progress made in connexion with the eradication of bovine tuberculosis. While as a profession taking our due share of credit for this achievement, we must acknowledge the excellent co-operation we have received, and are still receiving from the agricultural community; without that co-operation no scheme for eradication of prevention of disease can succeed. This fact must be borne in mind when other schemes are formulated. The goal of total eradication, which at one time seemed so remote, is now in sight. Seventy-one per cent of the herds in Great Britain are now free and it has been predicted that total eradication will be achieved within the next few years. The successful fight which is being waged against bovine tuberculosis is of great importance to the whole community, and a considerable diminution in non-pulmonary tuberculosis in human beings will undoubtedly result.

Vaccines and Disease Control

The development of new and effective vaccines has rendered possible the control of many diseases which were at one time the cause of immense losses. Noteworthy among these is that against *Brucella abortus* infection. The extensive use of Strain 19 consequent on the inauguration of the Calf Vaccination Scheme in 1944, which enables the farmer to have his calves vaccinated at low cost, has removed many of the terrors of this disease, and its extended use could in time pave the way for a policy of total eradication. The chief danger here appears to be that of complacency on the part of the present day stock-owners, many of whom have never experienced the ravages of an abortion storm in their herds. Similarly, the extended use of crystal violet vaccine against swine fever could go a long way towards ridding us of this particular menace. It does, however, appear necessary to emphasise to the farmer the prophylactic nature of this vaccine, and to make him realise that when swine fever exists on the premises it is generally too late to commence the use of vaccine.

The successful use of vaccines in connexion with some diseases must not lead us to accept a policy of vaccination as an answer to all disease problems; the advantages and disadvantages of the use of a

Our picture taken at the Eastern Counties Division "At Home" last Sunday evening shows, left to right, the President, Mr. E. R. Callender, Mrs. William Miller, The Earl De La Warr, Mrs. Callender, Mr. William Miller, President of the Eastern Counties Veterinary Society, and the Countess De La Warr.

vaccine must be carefully weighed by those responsible, before it is advocated; and if a specific disease can be more effectively controlled by means other than vaccination, then there is little point in changing the method of control. It is not so long ago that a small but very vocal minority was clamouring for a switch to vaccination in connexion with foot-and-mouth disease, despite the fact that this disease has been most effectively controlled by the slaughter policy for many years now. It is, however, gratifying to note that vaccination has been of considerable value when applied systematically in those countries where the disease is more or less endemic.

In this connexion it is not as widely known as it should be that one of the most valuable effects of vaccination will be to bring about a state of affairs when foot-and-mouth disease will be sufficiently reduced in incidence to be dealt with under a slaughter policy, and thereby eradicated from countries now suffering from its ravages.

Great steps have been taken by means of vaccines to control diseases of sheep which in the past caused tremendous losses, notably lamb dysentery, pulpy kidney, and louping ill. The increased interest taken by the veterinary profession in sheep disease problems during recent years has brought untold benefits to the sheep farmer who no longer regards losses from disease as inevitable. Internal parasites are, however, still responsible for considerable losses in terms of mutton, despite the greatly increased efficiency of present-day anthelmintics against some of the worm parasites, and it is a sobering thought that the liver fluke, with which we have so long been familiar, is still responsible for heavy financial losses.

Poultry

One cannot leave the sphere of preventive medicine without some reference to poultry. The small band of veterinary workers who have directed their attention to poultry diseases are entitled to our

gratitude and to that of the poultry industry for all that they have done. There are, however, many disease problems which remain unsolved, and unless the profession generally is prepared to take a more active interest in this aspect of our work we cannot make the progress which could and should be made. The present-day veterinary surgeon is well equipped with the aid of laboratory facilities, and the assistance of those within the profession who have made the study of poultry diseases their province, to handle poultry problems; and if he fails to exert himself in this direction he cannot complain if others, less qualified, endeavour to advise on disease problems to the detriment of the poultry industry. One is glad to note that the more recent graduates are taking a much greater interest in poultry diseases and that their advice is being more readily sought in this connexion. Those engaged in research upon problems connected with poultry disease have every reason to be proud of their achievements. To take but one example; the work done in veterinary laboratories in connexion with leucosis of poultry may yet throw light on the same disease in human subjects. As an instance of the parallel studies between veterinarians and doctors in this field, I would mention that at least one veterinary scientist has transformed his studies from poultry to human cancer research. However, in the field of poultry disease there is no room for complacency; much still remains to be done.

If we are to continue to progress we must have the utmost unity between all the various interests in the profession. In these days of specialisation, there is a tendency for sections to remain aloof from one another; while this is unavoidable to some degree, there must be at all times the closest co-operation between all the different bodies which comprise this great Association of ours. In a comparatively small profession such as our own, we must realise that each section is complementary to the others, and only harm would result to the whole veterinary body if we all went our separate ways in pursuance of interests concerned with our individual interests and careers. Only by presenting a united front and by proving ourselves capable of maintaining the highest possible standard of efficiency in all the duties we undertake can we hope to extend the sphere of our work and to make our maximum contribution to the community.

Contacts with Outside Bodies

I am sure we are all agreed as to the great importance of the friendly contacts we maintain with other bodies, and I am glad to be able to say that all those contacts continued to be strengthened during the past year. First, I would mention the cordial relationship which exists between our Association and the R.C.V.S. We have conferred together, I think I can safely say, upon every important matter affecting the well-being of the profession, and there is harmony in our views. We have been trying to give more space in our official journal to the activities of the Royal College, and for some time past, as you will have noticed, we have published in the *Supplement* full details of Council meetings. It is, of course, most necessary that our Association should be in constant intimate contact with the governing body of our profession, and it is a source of great pleasure to your Officers that this should continue to be so.

Other important contacts have been maintained, and include those with the National Farmer's Union, the medical profession, the animal feeding-stuffs trade, the pharmaceutical industry and—what is of great importance—the agricultural press, whose members we entertained informally to dinner earlier this year.

I must, of course, include in this list the Animal Health Division of the Ministry of Agriculture, Fisheries and Food. The fact that I am the second member of the veterinary staff of that Division to be elected as President of the British Veterinary Association in recent years is an indication of the happy relationship which exists between the Animal Health Division and the veterinary profession generally.

During my year of office tragic events in Hungary took place, and among the thousands of refugees who came to this country were a few veterinary surgeons. It was a privilege to make contact with them and to act as host at a small informal dinner party when we were able to offer a welcoming friendly hand. As you would know, there is a limit to our endeavours inasmuch as Hungarian veterinary qualifications are not registrable over here. However, we did our best for them on the personal and human side of things and they were very grateful. Most of their personal problems are, we are glad to note, well on the way to being solved.

Mention of troubles in other parts of the world recalls the difficulties brought about by the closing of the Suez Canal and the reintroduction of petrol rationing. As on former occasions, members of the Association very generously undertook to act as regional liaison officers between members of the profession and the rationing authorities. Their work has been of the greatest value; they succeeded in ironing out a great many difficulties and in helping members in practice to keep their cars in daily use. I take this opportunity of paying tribute to the excellent work they did.

The Animal Welfare Societies

At each Congress within recent years the President, in his annual address, has had something to say about relationship with the animal welfare societies. This year the most important event in that field was the emphatic rejection of the P.D.S.A. Bill by the House of Commons. As you know, this organisation introduced a private Bill which would have taken away from the Minister of Agriculture the right to withhold licences permitting non-veterinary staff in the P.D.S.A. employment to treat animals. Parliament turned it down without even going to a Division, and it is a source of satisfaction to us that the profession's undoubted right to be the statutory

(*Continued on page* 839)

A New Drug for the Treatment of Lungworms in Domestic Animals

BY

J. K. WALLEY

Imperial Chemical Industries Limited, Research Department, Pharmaceuticals Division

Part I

Introduction

IN many parts of the world parasitic diseases are responsible for great loss to the agricultural community. Although there are no complete figures for the incidence and distribution of lungworms in domestic animals, it is likely that they are equal in importance to gastro-intestinal worms. Foster (1954) estimates that helminth parasites cost the United States' live-stock industry about $400,000,000 annually. A large proportion of this is due to lungworm diseases, and Foster draws attention to the failure of known anthelmintics against extra-intestinal worms.

Lungworm disease in cattle and sheep has been recognised in all parts of Great Britain since before 1840 (White, 1842). That worms were the cause of the disease was recognised about 1850. The most harmful species of lungworms are those parasitic in sheep and cattle. They cause the disease called "husk," "hoose," "hoast," "Galar Cleibh," "Hach," or "Hych," being a parasitic bronchitis (phthisis pulmonalis verminalis). Work by Jarrett *et al.* (1954) has shown that in S. W. Scotland 50 per cent. of all bovine deaths in the period August to October in 1953 were attributable to lungworm infestation, and that it was the most important killing disease during these months. Pfizermaier (1951) estimates that 95 per cent. of the sheep of Württemburg are infested with lungworm. In Ankara (Turkey) abbatoirs, Gurlap (1952) found that 86 per cent. of 28,221 sheep harboured lungworms. Jarrett *et al.* (1955) of Glasgow in a small survey found adult lungworms in 31 per cent. of sheep lungs even in February. Our own work has led us to think that lungworms in sheep in Great Britain are at least as important as intestinal worms in causing death and unthriftiness in lambs during the period July to November.

Lungworms have been found in bacon pigs, live-weight 200 lb., to the extent of 15.7 per cent. (Dunn, 1954), 20.5 per cent. (Dunn *et al.*, 1955), and 22 per cent. (Walley, 1951), and in pork pigs (live-weight 100 lb.) to the extent of 47 per cent. (Walley, 1951). They are reported as widespread in young pigs in the U.S.A. (personal communications) and in Yugoslavia (Nevemio & Sibalic, 1953).

The lungworm diseases are generally more common in the young animal, but during the last 10 or 12 years have been seen to occur increasingly in older animals, *e.g.* in the milking cow. The total loss due to falling off in condition, loss of production, abortions, stunted growth, delayed deaths, and upset in herd replacement plans, is greater than the obvious loss due to the acute disease, *e.g.* Michel and Shand (1955) report an outbreak in a dairy herd where only 1 cow died but the loss of milk and cost of extra foodstuffs, etc., was £2,500.

The ill effects on the host are due not only to worms themselves, but also to the damage they cause which predisposes the host to bacterial and viral infection of the lung. The allergic reaction associated with infection and reinfection is also important. Michel (1954) has shown that a substantial proportion of "fog fever" cases is traceable to lungworm infection. Shope (1941, 1943) demonstrated that the larvae of the pig lungworm, *Metastrongylus apri*, may mechanically spread influenza of swine from infected to healthy pigs. It is possible that other virus diseases may be transferred in the same way.

The importance of lungworm disease is reflected in the many treatments which have been tried for it, *e.g.* for cattle and/or sheep limewater (Mayer, 1830), prussic acid (Williams, 1887), and turpentine with or without oils (Dickens, 1842; White, 1842; Dobson, 1872; Steel. 1887) have been given orally, and a great variety of substances intratracheally (Steel, 1887; Winslow, 1919; Hoare's Veterinary Therapeutics, 1942; Eveleth & Eveleth, 1942; Ortlepp, 1945; Montoya, 1946; Vingerhoet, 1950; Sonnet, 1950; Flovent, 1951; Behrens, 1951; Pfizermaier, 1951; Götye, 1952; Thomsen, 1953; Lungu *et al.*, 1953; Butorin, 1954; Stephanski, 1955; Sarwar & Rauf, 1956), etc. Parenteral injections have been tried, *e.g.* emetine hydrochloride intramuscularly (Turnova, 1949; Durbin, 1952), and diethylcarbamazine subcutaneously (Ozerskaya, 1955). Nasal installations have been used (White, 1842; Dobson, 1872; and Donigiewicz, 1954). Inhalations first advocated by Dobson (1872) have more recently (Enigk, 1055) been resurrected as a means of trying to bring anthelmintics, *e.g.* oil of chenopodium and santonin, into contact with the worms, and this treatment may be effective which, according to the reports on the diseases of live-stock (1944a, b, 1950), is more than can be said of intratracheal injections. Treatment in other species has shown the same lack of success, *e.g.* in pigs (Bable, 1949, Torozoma Vilas, 1951. Buck *et al.*, 1954), dogs (McGaughey, 1952), and cats (Sudduth, 1955).

The need for an effective, easily administered drug for lungworms caused us in these laboratories to search for one, and it is the purpose of this paper to describe the success we have achieved with cyanacethydrazide.*

* United Kingdom Patent Applications No. 28906/56, 37449/56, 37450/56, 37451/56, 37452/56, and 37453/56 pending. "Helmox" is the trade mark of Imperial Chemical Industries Limited for a formulation of cyanacethydrazide for the oral treatment of lungworm (Husk) in farm live-stock and "Dictycide" that of a similar formulation for injection and for pigs a special preparation. "Elimix," which also contains piperazine, is for the combined treatment of lungworms and roundworms.

Experimental Materials and Methods

Despite the normal host specificity of lungworms, it has been possible to develop experimental infections in guinea-pigs with *Dictyocaulus filaria, D. viviparus, Metastrongylus apri,* and *Muellerius capillaris* suitable for testing compounds. Full details of the screening tests done in collaboration with Mr. C. H. Vasey and Dr. N. Greenhalgh will be published shortly. For general screening we adopted the use of *D. filaria* in guinea-pigs.

Although the life-cycle of the worms is completed in the guinea-pig, to obtain sufficient larvae we used carrier goats or sheep and collected the larvae either by the usual faecal sack, or preferably, as we found later, by means of a polythene bag attached to a tracheotomy tube. In the latter case embryonated eggs and larvae were collected. The larvae were incubated to the infective stage before oral infection of guinea-pigs.

In the case of *M. apri,* eggs were collected from faeces from the pig-sty floor or from individual animals in a metabolism cage or from lungworms recovered at a bacon factory. Eggs and larvae were obtained and " stored " in earthworms for at least 2 years.

Compounds exhibiting activity in guinea-pigs were further tested against experimental infections in farm animals. In kids and lambs, 4,000 infective larvae of *D. filaria* were given to animals 2 to 6 months old, and 5,000 to those over 6 months old. In calves, 4,000 to 5,500 infective larvae of *D. viviparus* were used to infect each animal. When lambs or calves were known to have been previously slightly infected they were later reinfected by dosing orally with 50 per cent. more infective larvae than they were given before. Similar doses have been used to reinfect calves and lambs with previous natural infections. An estimated 4,000 Metastrongylus larvae were given as an infective dose to all pigs below 50 kg. liveweight.

As well as the artificial infections, sheep naturally infected with *D. filaria, Protostrongylus rufescens, Muellerius capillaris,* and *Neostrongylus linearis,* cattle naturally infected with *D. viviparus,* and pigs naturally infected with *Metastrongylus elongatus (apri)* were used for further trials.

The presence of lungworms in the lungs was diagnosed by faecal examination. In a heavy infestation eggs or larvae are found in every gramme of faeces examined. From field samples maximum samples of 400 g. were used as a routine to put through a Baermann apparatus to find larvae. After treatment as many animals as possible were killed for complete examination of the lungs, etc.

Results

Cyanacethydrazide—Its Properties

Cyanacethydrazide ($CNCH_2CONHNH_2$) is a white crystalline substance readily soluble in water. It has a somewhat bitter taste. When exposed to light it gradually turns a yellowish-brown. The solutions also become discoloured on exposure to light, more rapidly than the solid, and later a brown precipitate is formed. The solution has now been stabilised by my colleague, Dr. N. Greenhalgh. Cyanacethydrazide to which a stabiliser is added is readily soluble and in warm water a 25 per cent. w/v solution is quickly obtained. It was used for parenteral treatment. For oral administration the mixture of drug and stabiliser was used alone or mixed with diluents such as lactose, kaolin, or urea. Cyanacethydrazide is very soluble in urea solution.

The use of an oily (arachis oil) suspension of cyanacethydrazide did not prolong the activity of the compound, probably due to the marked solubility of cyanacethydrazide in water at body temperature.

Cyanacethydrazide—Anthelmintic Action

1. *General*

Observations in the experiments with guinea-pigs suggested that the lungworms are not killed *in situ* by cyanacethydrazide but somehow become dislodged, probably because they are damaged by the drug to the extent that they are unable to resist the ciliary movement in the air ways and are finally carried up the trachea. We regarded this as a most important point to check because clearly dead worms in the bronchi might cause severe reactions and it would be a most advantageous property of cyanacethydrazide if it caused the worms to be removed alive. Accordingly, experiments were done in calves, sheep, and goats fitted with tracheotomy tubes. These tubes were modifications of Degive's tube (Cadiot & Almy, 1901), and to their outer lip polythene bags were attached in order to collect any tracheal exudate or expelled worms, etc. (Fig. 1). We found that the compound did in fact cause the lungworms to be expelled alive. The times of expulsion and the state of worms at expulsion were observed. This procedure did not upset the animal markedly nor by itself did it cause expulsion of lungworms. Occasionally, in order to collect all the worms and exudate expelled the trachea anterior to the tracheotomy tube was occluded.

The use of tracheotomy tubes served not only to show the mechanics of the action of cyanacethydrazide but helped to decide how best to use the drug. Animals with tracheotomy tubes therefore feature largely in the experiments described below.

FIG. 1.—Collection of tracheal exudate and expelled worms after treatment of infected animal.

TABLE I

EXPULSION OF *Dictyocaulus viviparus* FROM THE LUNGS OF CALVES

Calf number	Live weight (kg.)	Each dose mg. per kg.	Number of doses given once daily on successive days	Route	Worms collected*				Worms at *P.M.*	
					1st 24 hours	2nd 24 hours	3rd 24 hours	4th 24 hours	Alive	Dead
510 E	55	25	4	Subc.	346(*a*)	152(*a*)	224	111	226	31
513 E	52	25	3	"	28	9	2	2	2	3
404 E	150	20	2	"	432	213	53	5	28	4
514 E	75	15	3	"	37	3	1	0	0	0
512	68	15	3	"	127	46	13	2	4	1
515 E	82	15	3	"	206	34	7	4	0	0
511 E	66	50	1	Oral	50	2	4	1	25	0
518 E	67	25	3	"	331	228	49	3	10	2
517	74	15	3	"	40	8	15	4	48	0
516 E	69	15	3	"	5(*a*)	115(*a*)	25	17	176	0
523 E	71	15	3	"	9	4	1	0	4	6
508 E	61	15	3(*b*)	Subc.	197	30	5	2	58	5
401	128	17·5	1	Oral	43	5	0	0	1	0
402	167	17·5	1	"	64	8	0	2	0	2
403	149	17·5	1	"	7	0	0	0	0	0
521	72	Control	—	—	0	4	0	3	803	44

* Only a proportion of those worms expelled were collected.
(*a*) These numbers are not complete as the force of the cough repeatedly burst the polythene bags during the first 48 hours.
(*b*) Doses at 12-hourly intervals.
(*c*) Experimentally infected.

2. *The Effect of Cyanacethydrazide against Lungworms in Cattle*

a. *Natural and Experimental Infections in Cattle Carrying Tracheotomy Tubes.* Detailed results are given in Table I.

The doses used were suggested by the work with guinea-pigs. Noteworthy facts are:—

(i) Oral treatment is almost as efficacious as subcutaneous treatment and unless contra-indicated this would be the preferred route.

(ii) Subcutaneous injection seems to cause a slight, transient pain but no reaction follows.

(iii) The majority of worms are removed within the first 24 hours in most cases and except in severe infections treatment on one day is probably sufficient as is shown by the results achieved with animals 401, 402, and 403.

(iv) There is little to be gained by increasing the dose above about 15 mg. per kg.

b. *Other Experimental Infections in Cattle.* (i) Six 2-month-old calves which had been experimentally infected each with 4,000 infective larvae of *D. viviparus* were received from Dr. McIntyre of Glasgow University. Twenty-four days after infection all were showing larvae in the faeces. On the 28th day they were used for experimental treatment. Two served as controls, a third was given 25 mg. per kg. × 3 subcutaneously, a fourth 15 mg. per kg. × 3 subcutaneously, the fifth received 25 mg. per kg. × 3 orally, and the last one 15 mg. per kg. × 3 orally. The subcutaneous injections were made as usual in the neck. The oral doses were given in the evening feed of milk substitute on 3 successive days. The results of the treatment are given in Table II (Experiment (i), calves 496-501).

The clinical improvement in the treated calves was obvious within a few days. Respirations became slower and easier. The exaggerated abdominal and head movement with each expiration ceased. *Post-mortem* examination showed that the lungs of treated animals were normal in appearance whilst the pulmonary oedema in the untreated calves was very marked. Fig. 2 shows the lungs from 2 of the calves. The lungs from the treated calf No. 501 which received 15 mg. per kg. orally for 3 days weighed exactly half that from the untreated calf No. 497. (The same effect is seen (Fig. 2) in twin kids that were treated—see below.) The improvement in these and similar animals, where there appears to have been little if any secondary infection, was always most marked. Secondary infection, however, calls for additional treatment (see below).

(ii) Four calves (Nos. 506-509, Table II) of low physique and known to be suffering from intestinal worms were infected each with 5,000 larvae of *D. viviparus*. All 4 developed very severe "husk" symptoms but all improved markedly after treatment.

FIG. 2.

Control calf No. 497. *P.M.* 1,567 worms.

Treated calf No. 501. 15 mg. per kg. x 3. S.C. *P.M.* 13 worms.

TABLE II

THE TREATMENT BY CYANACETHYDRAZIDE OF CALVES INFECTED WITH LUNGWORM, *Dictyocaulus viviparus* (EXPERIMENTAL INFECTION)

Experiment number	Calf number	Live weight kg.	Each dose mg. per kg.	Number of doses given once daily on successive days	Route	Larvae in the faeces	Killed days after 1st dose	Number of worms found in lungs	
								Alive	Dead
(i)	496	79	Control	—	—	Continuous	Not killed	—	—
	497	61	„	—	—	„	12	1,567	—
	498	65	25	3	Subc.	Intermittent after 7th day	14	32	8
	499	68	15	3	„	Absent from 7th day	14	13	0
	500	65	25	3	Oral in milk	„ 6th „	12	12	2
	501	71	15	3	„	Intermittent after 6th day	12	69	5
(ii)	506	98	Control	—	—	Continuous	8	986	17
	510	55	25	4	Subc.	„	8	226	31
	508	61	25	3*	„	Intermittent after 6th day	9	63	0
	509	120	20	3	Oral in 2 litres drinking water	Absent after 7th day	9	13	0
(ii)	525	215	Control	—	—	Continuous	7	183	7
	526	204	20	1	Oral mixture 10 per cent. cyanacethydrazide in urea add to corn ration	Absent after 5th day	7	0	0
	527	212	20	1					

* Doses given at 12-hour intervals.

The case history of 510 is given in detail for its interest. By the 23rd day after infection this animal had a complete anorexia, and stood with neck outstretched and tongue protruding; it had a sticky nasal discharge and an occasional harsh cough. After bouts of coughing it fell down. The respiration was 100-120 per minute. The temperature was 104° F. Auscultation revealed bronchial sounds and emphysema, pericardial fluid, and consolidation of the diaphragmatic lobes. The prognosis was therefore grave. A tracheotomy tube was inserted and the calf was given 1.3 g. of cyanacethydrazide (*i.e.* approximately 25 mg. per kg.) subcutaneously for 4 days. The total number of worms expelled is not known because the force of the cough burst the polythene bag repeatedly during the first 48 hours. In the 6-hour to 9-hour period 346 worms were collected. In the 24-hour to 30-hour period, 152 worms in 50 ml. of exudate were collected in an intact bag. After 48 hours the bags survived and 48 to 72 hours yielded 224 worms in 126 ml. of exudate, 72 to 96 hours 111 worms in 153 ml. of exudate, 96 to 120 hours 19 worms in 185 ml. of exudate, 120 to 144 hours 0 worms in 101 ml. of exudate, 124 to 144 hours 2 worms in 224 ml. of exudate, and 144 to 168 hours 0 worms in 175 ml. of exudate. Twenty-four hours after the first dose the calf showed signs of improvement and 48 hours later it was drinking milk and nosing the corn. Thereafter the clinical recovery was most marked. In 8 days' time it was obviously well on the way to normal. Auscultation, however, suggested that the left diaphragmatic lobe in particular was solid. The calf was killed and 257 worms were found in the air passages mostly imprisoned behind a cheesy exudate in the small bronchi especially in the left diaphragmatic lobe where there was a considerable area of bacterial pneumonia (pasteurella) and some oedema. Although the animal made remarkable progress, undoubtedly treatment for secondary infections would have resulted in a quicker and more complete recovery.

(iii) The efficacy of a single oral treatment in calves not severely infected is shown by the results with animals 526 and 527 (Table II).

(c) *Naturally Infected Cattle.* Fifty-six cattle (live-weight 165 to 480 kg.) naturally infected with *D. viviparus* were treated with cyanacethydrazide. They were of various breeds and their larval counts ranged from 3 to 100 larvae per gramme of faeces at the time of treatment.

In 4 yearlings and 2 calves it was shown that 7.5 mg. per kg. and 10 mg. per kg. was insufficient dosage. Twenty cattle dosed at higher levels and slaughtered for examination are included in Table III.

Comments on Table III

(i) The heifer in Trial 3 having 63 lungworms present after treatment had some evidence of a chronic pneumonia. (ii) The clinical response after treatment was noticeable in all the animals, with 1 exception. The exception was 1 of the 11 in trial number 4 which showed the typical dullness of chronic respiratory distress and was observed to have a pneumonic area on the left side. This resolved in less than 1 month. (iii) The treatment removed the *D. viviparus* from the " carrier " cattle (Trial 1). (iv) The dosing regimen used in the groups infected

TABLE III

THE TREATMENT WITH CYANACETHYDRAZIDE OF CATTLE INFECTED BY LUNGWORM, *Dictyocaulus viviparus*

Trial number	Number of animals	Average live-weight kg.	Each dose mg. per kg.	Number of doses given once daily on successive days	Route	Larvae in faeces after dosing	Killed days after 1st dosing	Average number of worms found in lungs	
								Mature	Immature in tissue
1 Light infestation "carriers"	3	420	5 g. (total)	1	Subc.	Absent 4th day	7	0	+ (in 1)
	3	400	5 g. (total)	3	Oral	„ „	7	2 (in 1)	—
	3	407	Control	—	—	Intermittent	7	32	+ (in 1)
2 Light infestation	3	225	15	3	Subc.	Absent 5th day	10	5 (in 1)	—
	4	230	17·5	3	Oral	„ 7th „	10	0	+ (in 1)
	3	240	Control	—	—	Continuous	10	163	+ (in 2)
3 Medium infestation	3	186	25	3	Subc.	Absent 6th day	7	0	+ + (in 2)
	1	200	20	3	„	Intermittent	6	63	+
	3(*a*)	251	15	3	„	„ after 6 days. Absent after 26 days	28	0	+ (in 1)
4 Medium infestation	11(*a*)	180–275	20	1	Subc.	Absent after 7 days. One + after 14 days. Absent after 26 days at least till 150 days	—	—	—
	4	180–210	Controls	—	—	Larvae in faeces for 48 days	—	—	—
5 Medium - heavy infestation	9	280–410	15 to max. 5 g.	(*b*)	Subc.	Absent after 12th day	—	—	—
6 Medium infestation	10	160–240	17·5	(*b*)	Oral	Absent after 14th day	—	—	—

(*a*) Dosing repeated 21 days after first treatment.
(*b*) Dosing made only on day disease confirmed (Day 1) and on Days 2, 10, 11 and 21.

naturally on pasture (Trials 4, 5, and 6) controlled the disease by removing the worms as they developed in the air passages. (v) In Trial 5, there was no interference with pregnancies of 2, 5, 6, or 8 month's duration. (vi) The drug had no effect on migrating *Hypoderma bovis* larvae in 2 cattle in Trial No. 6.

3. *The Effect of Cyanacethydrazide against Lungworms in Sheep*

Experimental work with sheep and goats has been primarily confined to *D. filaria* and *M. capillaris* with a small number of animals infected with *P. rufescens* and *N. linearis*. The disease caused by *D. filaria* in sheep and goats is essentially similar to that caused by *D. viviparus* in cattle, *i.e.* it is a "husk." To a varying degree the respiration becomes difficult, the rate at first increasing, the animal assumes a characteristic posture and may have a nasal discharge, early diarrhoea, dehydration, emaciation, anaemia, and later obvious heart dilatation and circulatory difficulties. The diseases caused by Protostrongylus, Muellerius, and Neostrongylus are similar but less severe. The pathology of the lung tissue due to the presence of migrating larvae, adults, eggs, and first-stage larvae in the air ways is essentially the same as described in detail by the Glasgow workers for cattle infested with *D. viviparus* (Jarrett & Urquhart, 1954).

a. *Natural and Experimental Infections in Sheep Carrying Tracheotomy Tubes.* The activity of cyanacethydrazide in expelling *D. filaria*, *P. rufescens*, and those *M. capillaris* and *N. linearis* that were present in the air passages was shown in the 14 lambs recorded in Table IV.

The general pattern of the results for sheep is very similar to that recorded for cattle in Table I and essentially similar conclusions may be drawn.

b. *Other Experimental Infections in Sheep.* A single lamb, No. 147, live-weight 28 kg., was infected with 4,000 infective larvae of *D. filaria*. Thirteen days later it was given cyanacethydrazide at the rate of 25 mg. per kg. subcutaneously for 3 successive days and on the fourth it was killed, *i.e.* the 16th day of its infection. Detailed examination of the air passages and of the washings from them did not reveal any immature or fifth-stage larvae. Squash preparations and a Baermann examination of minced lung did, however, yield fifth-stage larvae. From this it would appear that cyanacethydrazide will cause the expulsion of immature Dictyocaulus actually in the air passage early in an initial infection. Whether or not this activity will save the lives of animals affected by the hyperacute syndrome (Michel, 1954) is not yet known but a severe outbreak in the flock (or herd) might well be prevented by treatment at the time early symptoms are seen.

TABLE IV

EXPULSION OF FILARIA FROM THE LUNGS OF LAMBS AND KIDS

Animal number	Live-weight (kg.)	Dose mg. per kg.	Number of doses once daily on successive days	Route of dose	Worms collected				Worms at *P.M.* (5th day)	
					1st 24 hours	2nd 24 hours	3rd 24 hours	4th 24 hours	Alive	Dead
Lambs										
226	24	25	3	Subc.	99(*b*)	23	5	0	5(*d*)	3(*e*)
103(*a*)	22	25	3	,,	284(*b*, *c*)	72	12	2	3(*d*)	1
282	35	15	3	,,	102	78	4	5	1	0(*e*)
245	19	15	3	,,	260(*b*)	217(*b*)	92	34	87	4
271	25	15	3	,,	117	14	3	0	6(*d*)	0
225	22	15	2	,,	148(*c*)	100(*c*)	6(*c*)	2	26(*d*)	7(*e*)
104	30	15	2	,,	95	29	4	0	5	3
229	25	25	3	Oral	36	6	0	0	0	0(*e*)
236	20	20	3	,,	78	257(*c*)	116	6	2(*d*)	5
270	24	15	3	,,	105	235(*c*)	74	10	33	7(*e*)
285	24	15	3	,,	186(*b*)	104(*b*)	22	10	0	0(*e*)
193	23	15	3	,,	42	34	11	10	4(*d*)	0
205(*a*)	25	15	3	,,	172	307	143	7	7	2(*e*)
158	30	20	2	Intratracheal	48	57	10	2	36	5
Kid										
54	23	15	3	Subc.	147	156	54	6	3	0

(*a*) In this lamb all the worms expelled were collected by blocking the trachea anterior to the tracheal tube ; in all other cases only a proportion were collected in the polythene sac.

(*b*) In these cases *rufescens* were also found in the sacs *viz.*: No. 103 had 17 worms, No. 245 had 22 worms, No. 226 had 5 worms and No. 285 had 6 worms.

(*c*) In these animals *capillaris* were also found in the sacs *viz.* : No. 103 had 46 worms, No. 225 had 33 worms, No. 236 had 6 worms. No. 103 and No. 225 each had 6 *linearis*.

(*d*) Live *capillaris* were found in the lungs at *post mortem*.

(*e*) Immature *filaria* were found in the tissue of the lungs.

TABLE V

THE TREATMENT WITH CYANACETHYDRAZIDE OF SHEEP INFECTED WITH FILARIA

Plot number	Number of lambs	Average live-weight (kg.)	Each dose mg. per kg.	Number of doses given once daily on successive days	Route	Larvae in faeces after dosing	Killed days after 1st dose	Average number of worms found in lungs	
								Mature	Immature in tissue
3	5	32	25	3	Subc.	Absent after 6th day	10	8	+
	4	32	Control	—	—	Continuous	10	199	+
1	5	33	15	3	Subc.	Absent in 4 after 6th day, 1 intermittent to p.m.	6	1	+
	3	29	Control	—	—	Continuous	6	121	+
9A	5	22	20	3	Oral	Intermittent	6	19	+
	5	21	Control	—	—	Continuous	6	247	+
9B(*a*)	5	19	175	(*b*)	Oral	Intermittent	22	3 (in 1)	+
	5	20	Control	—	—	Continuous	22	115	+ +
9C	8	23	15	(*b*)	Subc.	Absent after 24 days for at least 120 days	—	—	—
	3	24	Control	—	—	Larvae in faeces for 53 days	—	—	—
10	1 ewe	20	20	3	Subc.	1,000 larvae per g. on day 1 of dosing	4	60	—

(*a*) The average live-weight gain in the 5 treated lambs was 3 lb. (1·4 kg.) more than in untreated animals in the 21 days after the 1st dose.

(*b*) The 1st dose was given on the 23rd day after the sheep were put to graze on a pasture heavily infested with lungworms. Dosing was repeated on the 24th, 30th, 31st, 41st and 42nd days. Autopsies, where done, were made on day 44.

c. *Naturally Infected Sheep*. The effect of treatment with cyanacethydrazide on sheep infected naturally with *D. filaria* is shown in Table V.

The results in Table V give ample confirmation of those listed in Table IV. Treated sheep were left with far fewer worms than those untreated, and it would appear that the oral dose need not exceed 17·5 mg. per kg. and the subcutaneous dose need not exceed 15 mg. per kg. The pastures on which the sheep were grazed were also heavily infested with *M. capillaris* larvae but there was no apparent action against the worms in the substance of the lung.

4. *The Effect of Cyanacethydrazide against Lungworms in Goats*

The expulsion of *D. filaria* from a kid after treatment with cyanacethydrazide at the rate of 15 mg. per kg. bodyweight subcutaneously is shown in Table IV. After treatment this kid had only three worms left in its lungs whilst the control twin kid had 1,166 worms.

Table VI gives the results of treating a number of goats with cyanacethydrazide both subcutaneously and orally at various dose levels.

This work was done early in the investigation which is why the doses are so scattered. The results with kid 54 (Tables IV and VI) suggest that doses similar to those for cattle and sheep would be efficacious. In all cases treatment was given 24 to 30 days after infection when there were larvae in the faeces and the animals showed typical symptoms of early acute " husk."

5. *The Effect of Cyanacethydrazide against Lungworms in Pigs*

The pigs reported on in this paper were free of virus pneumonia and were purchased from farms considered to be free of that disease. The results refer to pigs of weights most likely to be carrying a heavy infestation in the field.

Table VII shows the results of treating pigs, infected naturally and artificially with *M. apri*, by subcutaneous injections of cyanacethydrazide, or mixing it in the food with an ascaricide for pigs infected by both the lungworm and *Ascaris lumbricoides*.

The results in Table VII show that cyanacethydrazide at a rate of 15 mg. per kg. or over is effective in removing lungworms from pigs. The rapid disappearance of the ova from the faeces and the *post-mortem* findings indicated that some 97 per cent. of the worms were eliminated. The lungs of the treated pigs were smaller, less oedematous, and had fewer areas of emphysema and the mucous membranes were clear and free of exudate.

There is little evidence to hand of the influence of lungworm infection on growth rate in pigs. In an experiment where 2 groups of 8 pigs were maintained under the same conditions, except that 1 group was infected with *M. apri* larvae at 9 weeks of age, the infected group took 9 days longer to reach an average of 120 lb. live-weight (Walley, 1951). It is of note that the 2 control pigs in Trial No. 2, Table VII had a marked chronic cough, maintained a higher respiratory rate than the treated pigs, and although they had a weight advantage (average 3 kg.) on the day of first treatment the treated pigs reached an average of 50 kg. (110 lb.) live-weight 3 days before them. (See Table VII overleaf.)

6. *The Effect of Cyanacethydrazide against Gape-worms in Poultry*

The results of a preliminary experiment suggest that cyanacethydrazide has some action against *Syngamus trachea* in chickens but verification is needed and the work is continuing.

TABLE VI

THE TREATMENT OF GOATS INFECTED WITH FILARIA BY CYANACETHYDRAZIDE (EXPERIMENTAL INFECTION)

Experiment number	Goat number	Live weight (kg.)	Each dose mg. per kg.	Number of doses once daily on successive days	Route of dose	Killed days after first dose	Number of worms found
1(*a*)	42	11	100	1	Subc.	Died 2½ hours	2,043
	41	13	50	1	"	6 days	0
	40	11	25	1	"	6 "	206
2	52	14	Control	—	—	7 days	2,846
	51	12	25	3	Subc.	7 "	87
	50	14	10	3	"	7 "	741
3	43	22	Control	—	—	7 days	1,624
4	4	28	25	3	Oral	7 "	0
	48	19	25	3	Subc.	7 "	1
	49	21	100	1	Oral	7 "	0
4(*b*)	36	34	50	1	Subc.	Dead in 13 hours	1,826
	37	46	50	1	"	Dead in 10 hours	1,273
	38	43	50	1	Oral	6 days	5
	39	60	25	1	Subc.	7 "	2
5	53	27	Control	—	—	5 days	1,166
	54	23	15	3	Subc.	5 "	3

(*a*) Kid No. 42 acts as a control for Nos. 40 and 41.

(*b*) The 4 goats in section 4 were not all treated at the same time.

In experiments 1 and 2, triplet kids were used and in experiment 3 two sets of twins. Each had been given 4,000 infective larvae orally.

Experiment No. 4 is essentially a collection of 4 goats from the same herd and infected with 6,000 infective larvae *orally*.

TABLE VII
THE TREATMENT WITH CYANAZETHYDRAZIDE OF PIGS INFECTED WITH *Metastrongylus apri*

Trial number	Treatment	Dose (mg. per kg.)	Number of pigs	Average live-weight (kg.)	Average number of worms at *P.M.* 7 days after treatment	
					M. apri	*Ascaris lumbricoides*
1						
Artificial infection with *M. apri*	Cyanacethydrazide	50 subc. once only	1	19	0	—
	"	25 subc. on 3 successive days	3	21	12	—
	"	20 subc. on 3 successive days	4	20	0	—
	"	15 subc. on 3 successive days	3	22	3	—
	Control	—	2	21	471	—
2*						
Natural infection housed 10 days before dosing	cyanacethydrazide	50 subc. once only	1	16	No ova* after 5 days	—
	"	25 subc. on 3 successive days	3	21	No ova after 7 days	—
	"	15 subc. on 3 successive days	2	20	"	—
	cyanacethydrazide as 10 per cent. soluble in arachis oil	15 subc. on 3 successive days	2	20	"	—
	Control	—	2	24	Ova for at least 28 days	—
3						
Artificial infection *M. apri*	cyanacethydrazide and piperazine diacetate ratio 1 : 6 by weight	115 once daily in food for 3 days	4	50	0	0 (45 ascaris collected in faeces)
	cyanacethydrazide and 1-diethyl-carbamyl 4-methyl piperazine ratio 3 : 5 by weight	40 once daily in food for 3 days	2	52	2	1 (19 ascaris collected in faeces)
	Control	—	2	52	242	14
4*						
Natural infection *M. apri*	cyanacethydrazide	30 subc. for 3 successive days	10	15	No ova after 5 days	—

*Not killed until bacon weight.

The Use of Cyanacethydrazide in Combination with Other Drugs

In our experience the broncho-pneumonia caused by the present of lungworms has been complicated by secondary infections of the lung in about 15 per cent. of the cases. In the field, lungworm infestation in animals will also be concurrent with infestation by intestinal worms, and often, especially in younger animals, by coccidia.

The use of cyanacethydrazide in combination with anthelmintics, coccidiostats, and antibacterial agents may therefore be important, and experiments were done to determine that no interference of action between the different drugs would be encountered. As the results given below show, no interference was found and so dual treatments may be made when it is convenient to do so.

Cyanacethydrazide with Sulphadimidine

The combined use of sulphadimidine and cyanacethydrazide, both at therapeutic dose levels, is shown in Table VIII. For oral administration the 2 compounds were mixed into water and used as a drench. For subcutaneous administration each was injected independently. Sulphadimidine was used as "Sulphamezathine"* 33⅓ per cent. sterile solution. The results show that the independent action of the 2 drugs was maintained. The clinical improvement in the treated animals was most marked.

Cyanacethydrazide with Phenothiazine

The combined use and effectiveness of cyanacethydrazide and phenothiazine (as "Phenovis"*) has been demonstrated and the results are given in Table IX.

Comments on Table IX

(i) The result with the heavily infested lambs which were very dehydrated, anaemic, emaciated, and extremely weak—in fact 2 were recumbent—indicates that removal of the worms in itself is not sufficient to ensure recovery in severely affected animals. (ii) In these experiments phenothiazine was shown to kill first-stage *D. viviparus* and *D. filaria* larvae whilst in contact with them in the gut or in the faeces. The period over which action was noted varied with the rate of passage of the phenothiazine through the gut, *e.g.* 12 to 36 hours after a single

* Trade marks of Imperial Chemical Industries Limited.

dose. This action of phenothiazine is important because when animals are treated with cyanacethydrazide large numbers of eggs and larvae are released into the gut from the lungworms when these are expelled from the air passages and they would normally pass out to become infective on the pasture or bedding. (iii) In Experiment L2 all the lambs continued to graze the infected pasture for 6 weeks after treatment. There was no significant live-weight gain in the treated over the untreated animals. In contrast, in Experiment L3 the lambs were treated early before the chronic pulmonary changes had occurred and given repeated doses to prevent the establishment of a heavy infestation, and they gained an average of 3 kg. live-weight more than the control animals by 5½ months of age.

Cyanacethydrazide with Phenothiazine and Hexachlorethane

The oral dosing of a group of 5 lambs, average live-weight 22 kg., with a freshly prepared mixture of 5. g. " Phenovis " plus 3 g. " Avlothane "* and 0.4 g. (approx. 18 mg. per kg.) cyanacethydrazide daily for 3 days reduced the intestinal nematode egg

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TABLE VIII
THE COMBINED USE OF CYANACETHYDRAZIDE AND " SULPHAMEZATHINE " IN ANIMALS

Animal	Number	Average Live-weight kg.	Concurrent Infection	Dose: Cyanacet-hydrazide mg. per kg.	Dose: Sulpha-mezathine mg. per kg.	Route on 3 successive days	Average Parasites per g. of faeces: 1st day of dosing: Lung-worm larvae	1st day of dosing: (a) o.o. cysts	6 days later: Lung-worm larvae	6 days later: o.o. cysts
Calf	1	157	*Eimeria Zurnii* (acute coccidiosis)	20	120	Oral	120	200,000	0	5,000
Lambs	4	12	*E. avloingi*	15	150	Oral	10–100	40,000	6	12,000
	4	12	*E. faunei*	Control	Control	—	10–100	42,000	5–90	37,000
			E. intracator (sub-acute coccidiosis)							
Pig	1	18	*E. debliecki*	20	150	Subc.	25	125,000	2	4,600
	2	20	*E. scabra* (sub-acute coccidiosis)	Control	Control	—	38	40,000	26	21,500
Calves	2	118	Enteritis with intense scour	15	120	Oral	10–20	—	0	—
Calves	2	94	Pasteurella pneumonia	20	120	Subc.	60	—	+	—
	2(*b*)	98		Control	Control	—	74	—	35	—

(*a*) *Viviparus* in calves, *filaria* in lambs and *apri* in pigs. (*b*) One calf died on 6th day ++ pasteurella, 526 *viviparus*.

TABLE IX
THE COMBINED USE OF CYANACETHYDRAZIDE AGAINST LUNGWORMS AND " PHENOVIS " AGAINST INTESTINAL WORMS IN CATTLE AND SHEEP

Animal	Number	Average live-weight kg.	Dose: Cyanacet-hydrazide mg. per kg.	Dose: "Phenovis" g.	Oral dose on succes-sive days	Average Parasites per g. of faeces: 1st day of dosing: Lung-worm larvae	1st day of dosing: Nema-tode eggs*	7 days later: Lung-worm larvae	7 days later: Nema-tode eggs	Parasites at *P.M.* 8 days after 1st dose: Lung-worm	Parasites at *P.M.* 8 days after 1st dose: Nema-todes
Cattle C1.	2(*a*)	167	15	15	3	200	600	0	50	—	—
"	2(*a*)	170	17·5	40	1	5	300	0(*b*)	20	—	—
Cattle C2	3	230	20	40	1	52	330	3	40	14	1,050
	3	250	Control	Control	—	47	360	42	410	376	2,743
Lambs L1.	1	24	15	5	3	200	10,000	0	300	0	—
	1	25	50	25	1	200	14,000	25	2,700	47(*b*)	6,500
	3	20	15	5	3	400	18,000	<1	300	84(*c*)	5,780
	2	22	Control	Control	—	340	21,500	270	26,000	876(*c*)	21,400
Lambs L2.	5	30	50	20	1	340	3,820	14	110	—	—
	5	30	Control	Control	—	310	4,020	190	3,850	—	—
Lambs L3.	10(*d*)		20	20	1					—	—
	10		Control	Control	—					—	—

*Mixed infection, Haemonchus, Ostertagia, Trichostrongylus, Nematodirus.
(*a*) Same 2 heifers. Larvae again in faeces 10 days after 1st treatment. Second treatment given on the 18th day. No larvae found after 24th day for at least 64 days.
(*b*) Died on 7th day. Most of the 47 worms imprisoned behind plugs blocking bronchi of the caudal lobe.
(*c*) Two died, 1 on 6th, 1 on 7th day. Lambs (*b*) and (*c*) groups *in extremis* when treated.
(*d*) Dosed at 2 months, 3 months, 4 months and 5 months of age whilst on heavily infected pasture.

A Transmissible Polio-encephalomyelitis of Pigs (Talfan Disease)

BY
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FOR some years it has been evident that there occurs, in Great Britain, a non-suppurative polio-encephalomyelitis of young pigs which differs from any of the diseases so far recognised in the country. The purpose of this paper is to record the clinical and pathological manifestations of the disease, provisionally named Talfan Disease, and to describe a series of experiments demonstrating its transmissible nature.

The Disease in the Field

I. *Clinical Aspects*

The following summaries of outbreaks give a picture of the usual epidemiological and clinical features of the disease. In each outbreak the diagnosis was established by pathological examination.

Outbreak I (June, 1952, D. Buntain, then A.V.I.O. Cambridge).

Piglets were affected in 2 out of 7 litters running in contact. The only signs were inco-ordination with inability to stand on the hind legs. Appetite was not affected and the piglets were in good condition. No further information is available about this outbreak.

Outbreak II (April, 1955, E. A. Gibson, A.V.I.O. Cambridge).

Over the course of about 6 months, commencing about September, 1954, 2 or 3 pigs out of every litter in a herd of Large White pigs showed nervous signs. The earliest cases were febrile and responded to penicillin; possibly, these were cases of streptococcal meningitis. Subsequent cases were afebrile, and did not respond to penicillin. Crystal violet vaccine had not been used in the herd for the past year, but the breeding stock were the vaccinated survivors of an outbreak of swine fever 2 years previously.

The usual age of onset of the disease was 6 to 10 weeks. Affected pigs were at first unsteady if chased. Later, they suddenly became recumbent. There was some initial excitement, but soon the pigs rested quietly on their briskets, with their legs tucked under them. Appetite remained normal, and none died. The animals improved slowly over the next 4 to 6 weeks, but the legs tended to be deformed. A few of the pigs that had not shown evidence of paresis carried their heads on one side. All the pigs were in poor condition, with "rough" curly coats. Coughing was prevalent, and *post-mortem* examination showed the presence of a pneumonitis resembling the so-called virus pneumonia of pigs (V.P.P.).

All were second litters, and got by the same boar which was believed not to be related to the dams. One sow had previously had an unaffected litter by the same boar. The sows did not run out. Three diets had been used, 2 proprietary sow and weaner feeds and 1 home grown mixture. Cases occurred on all 3 diets.

One litter, affected at 6 weeks of age, was treated with cod-liver oil. The treated pigs appeared to recover more quickly and their legs did not become deformed.

A New Drug for the Treatment of Lungworms in Domestic Animals—*Continued.*

count from 10,500 e.p.g. to 100 e.p.g., and the *D. filaria* larvae count from 300 larvae per g. to 0 in 7 days. There was no observable change in Muellerius or Neostrongylus larvae output. The number of *Fasciola hepatica* eggs per g. of faeces increased from 40 to 110 e.p.g., a not unusual result following the expulsion of flukes from the bile ducts and the consequent sudden release of eggs causing a temporary rise in egg count.

Cyanacethydrazide with Piperazine

From experience in England and Wales it is clear that *M. apri* and *A. lumbricoides* will often be present together in the same pigs, especially in pigs less than 50 kg. (5½ score lb.) live-weight. Treatment for lungworms should therefore be used in conjunction with an effective ascaricide. Two examples of a successful combined treatment are given in Table VII. Mixtures of (a) cyanacethydrazide and tripiperazine dicitrate in the ratio 1:6 parts by weight and (b) cyanacethydrazide and 1-diethylcarbamyl-4-methylpiperazine in the ratio of 3:5 parts by weight removed the majority of the worms of both species. In both experiments the lots were fed as a group and the anthelmintics were mixed into the food. The advantage of both active agents being readily soluble in water leads to ease of mixing for group feeding and medication. In trial No. 3 (Table VII) the 6 treated pigs showed clinical improvement within a few days of the first dose. There was a marked slowing and improved regularity of the respiratory rate. Within 10 days the average respiratory rate of the treated pigs had fallen from an average of 68 per minute to 35 per minute. The controls had remained at 50 to 62 per minute.

(*This paper will be concluded next week*)

In April, 1955, the remaining affected pigs were slaughtered. No further cases until about August, 1956. Then 1, or sometimes 2 cases, showing similar nervous symptoms, were seen in occasional litters. The same boar was in use throughout this period, and the breeding sows were still kept indoors.

Outbreak III (December, 1955, A. Ratter, then V.I.O. Cheshire).

This was a herd of 40 Large White × Wessex sows. Initially 5 piglets from 4 litters became affected at about 3 weeks of age. The piglets first showed a vague weakness of the legs, progressing to a definite inco-ordination. They showed a tendency to walk backwards, and soon became recumbent on their sides. Appetite was reduced. Two died and 1 was killed for examination. One which was subsequently killed for examination, and 1 which was hand fed, recovered. A week later, 2 more pigs from 1 of the litters became affected, and 1 was killed for examination. The other showed marked inco-ordination for some time but was brought to the Weybridge laboratory where it finally recovered from the nervous disease though it failed to grow properly. One further suspected case occurred in December, 1956, but was not available for pathological examination.

Outbreak IV (March, 1956, C. V. Watkins, A.V.I.O. Exeter).

The disease appeared in a small herd made up of 3 gilts, 3 sows and their followers. About a third of 2 litters was involved, the progeny of Landrace sisters mated to the same boar. The piglets throve up to about 3 weeks of age when a number showed leg weakness followed by paralysis, first of the fore and later of the hind limbs. Sensation in the legs appeared to be diminished; pricking produced a simple reflex withdrawal but no obvious annoyance on the part of the piglet. Appetite remained good and the piglets would crawl to the sow to suckle. Body temperatures were within the normal range throughout the clinical disease. Two piglets died about 10 days after the onset of disease, but the survivors made complete recoveries and grew well. No vaccination was practised in the herd, and there was no evidence of ticks on the farm.

Outbreak V (June, 1956, G. F. Kershaw, A.V.I.O. Aberystwyth).

The disease broke out on a Ministry of Agriculture experimental farm which carried some 200 Large White and Welsh pigs, of which about half were used for feeding and husbandry experiments. The disease history on the farm had shown a variety of conditions to be endemic and frequent purchases were being made.

A system necessitating the yarding of some sows throughout pregnancy was in progress to develop a "virus-pneumonia-free" herd and segregation had been carried out on the assumption that certain sows were free from that condition. No anthelminthic treatment was carried out on any of the sows on the farm prior to farrowing. No iron therapy was practised on the young piglets, but sterilised soil was put in the pens. Crystal violet swine fever vaccine was given as a rule to piglets at about 4 to 5 weeks of age, but in 1 of the reported cases, no vaccine had been given to the piglet. The diets of both the piglets and the sows did not show any unusual features and were made up from foodstuffs grown on the farm. No extra minerals were given at any age. In certain cases the piglets were weaned at 2 weeks, housed in groups of 1 or 2 litters and fed on a proprietary foodstuff.

Four batches of piglets were affected with the condition which was characterised by initial "fits" followed in some cases by death, but more usually by inco-ordination. Except in the case of piglets dying in the early stages, recovery generally took place, although in some animals inco-ordination persisted.

Group A. Two pigs died and 4 others were affected in a group consisting of 2 litters. Their mothers had not been out at grass and the piglets were weaned at 2 weeks and fed on a proprietary product. Three days prior to signs of illness, the batch had been inoculated with crystal violet swine fever vaccine. Two of the pigs, 1 alive and the other dead, were examined at the Aberystwyth laboratory and, although some congestion was present in the lungs and meninges of the brain, no other abnormal findings were present and a cultural examination was negative. The worst affected piglet from the batch was killed and the brain and spinal cord sent to Weybridge for histological examination. The rest of these affected piglets improved considerably in condition and after a further 2 months it was difficult to detect any abnormal signs.

Group B. The disease occurred in the litter of a sow which had been out at grass for a limited period during pregnancy, but for the majority of the time was kept indoors. The piglets had sucked the sow for the normal period and had not been vaccinated. In this group "fits" were a characteristic feature of the clinical picture but only occurred in the early stages of the disease. These initial fits were violent and in some cases the piglets would be on their backs, apparently unconscious; some died during the "fits." However, the majority recovered but suffered from residual inco-ordination. *Post-mortem* examination once more showed no significant macroscopic lesions and cultural findings were negative.

Group C. In this instance, 5 piglets were affected and 1 death occurred. The sows were at grass. Crystal violet vaccination had been carried out a week prior to the onset of typical signs. Swine erysipelas serum had been given to all the piglets without visible effect. No *post-mortem* examination was carried out on any of these animals.

Group D. Only 1 piglet appeared to be affected in this litter. The sow, which had been yarded during pregnancy, developed an arthritic condition and lost her milk a few days after farrowing with the result that 7 of her litter died and only 4 piglets remained. These were inoculated with crystal violet swine fever vaccine 3 days before the clinical signs developed. These signs were again typical in that "fits" developed in the first instance, and were followed by inco-ordination. This piglet furnished the material on which the experimental investigations were based.

Outbreak VI (September, 1956, E. A. Gibson, A.V.I.O., Cambridge).

In this outbreak the disease appeared in a self-contained pedigree herd of Large Black pigs. The last purchase had been made in November, 1955, but animals were sent to 5 major shows during the summer of 1956, the last being in mid July.

During a period of 4 weeks, commencing August 14th, seven 7- to 8-month-old gilts, an 8-month-old boar and a 6-month-old hog developed posterior weakness and inco-ordination. None of the younger pigs on the farm was affected, nor were any of the older breeding stock. The groups of pigs involved had been vaccinated with crystal violet swine fever vaccine at about 6 weeks of age. The 7 affected gilts came from a batch of 55, kept as 4 groups. In the first group, 4 out of 13 gilts were affected. Three became recumbent, with flaccid paralysis of the hind limbs, while the fourth showed posterior inco-ordination but made a partial recovery. In the second group, one of 21 gilts became intermittently recumbent but could progress for short distances with the hind legs flexed, giving a rabbit-like gait. In the third and fourth groups inco-ordination and recumbency were shown by one of 15 and one of 6 gilts, respectively. The 8-month-old boar made a partial recovery after developing symptoms of posterior weakness. The hog pig, one of a batch of 10, showed marked ataxia, but did not become recumbent.

The body temperatures of the clinically affected pigs were within the normal range in all cases, and appetites remained normal. The time taken for the nervous symptoms to develop varied from 1 to 6 days. None of the 9 affected pigs died, but during the course of the outbreak 3 were slaughtered when completely paralysed. All 3 passed an inspection for fitness for human consumption. A fourth paralysed pig was submitted to Cambridge Veterinary Investigation Centre for laboratory examination. The central nervous system showed lesions indistinguishable from those of Talfan disease, and the authors consider that these lesions, together with the typical clinical picture, justify a diagnosis of Talfan disease, even with this unusual age incidence.

The remaining clinically affected animals were slaughtered about 1 month after the onset of the outbreak. No subsequent cases have been reported from this herd.

II. Histopathology of the naturally occurring disease

Wherever possible standard sections of the central nervous system were taken as follows:—

1. Transverse section through one cerebral hemisphere at the level of the corpus striatum.
2. Transverse section through the anterior corpora quadrigemina.
3. Transverse section through the cerebellum, or longitudinal section of the vermis.
4. Transverse section through the medulla oblongata at the level of the obex.
5. Transverse sections of spinal cord and dorsal root ganglia at various levels.

All sections were stained with haematoxylin and eosin, some were also stained by the Loyez Iron-Alum-Haematoxylin method, Marchi's method for degenerating myelin and Glee's modification.

Spinal Cord. Ten of the 11 spinal cords examined revealed lesions believed to be typical of the disease under discussion. The most severe damage was always found in the ventral horns, in the form of considerable perivascular round cell infiltration and severe focal and diffuse gliosis; similar changes were often present, though to a much lesser degree, in the dorsal horns. Some of the large neurones showed chromatolysis nuclear margination, karyolysis and some degree of satellitosis; neuronophagia was not conspicuous, but in the more chronic cases there was an apparent reduction in the number of motor neurones. Perivascular round cell infiltration and, more rarely, scattered foci of gliosis were sometimes present in the white matter, but the inflammatory reaction was always much less marked than in the grey. Sections stained by haematoxylin and eosin often showed an apparent rarefaction of the myelin in the dorsal funiculi. By Marchi's and Glee's methods degenerating myelin could be demonstrated scattered through the white matter, but usually bilaterally symmetrical and most concentrated in the dorsal funiculi. This process was usually fairly mild, but on two occasions it was developed to a marked degree and also involved the spinal nerve roots. By Loyez's method, the spinal nerve roots frequently showed considerable rarefaction of myelin. It is interesting to speculate whether Talfan disease may not be responsible for the sporadic outbreaks of ataxia with mild spinal demyelination frequently seen in older pigs. In the acute cases, the degree of demyelination may well be related to the length of time between the onset of signs and the death of the animal.

Dorsal Root Ganglia. Again the characteristic lesion was absent in only one case where dorsal root ganglia were available for examination; this was the same case which failed to show unequivocal lesions in the spinal cord. In all other ganglia examined there had been severe infiltration with cells of the lymphocyte and macrophage series, usually accompanied by a much smaller number of eosinophiles. Scattered neurones occasionally showed slight cytoplasmic vacuolation and nuclear margination and in some ganglion cells there was swelling of the nucleolus or shrinkage and crenation of the nucleus. Neuronophagia was rarely seen. The round-cell infiltration usually involved the periphery of the ganglion and the adjacent parts of the nerve root, but at a little distance the radiculitis, though present, became much milder.

The lesions of the spinal cord and dorsal root ganglia were most developed in the lumbar region and became progressively less in the more anterior parts. Lymphocytic infiltration of the spinal leptomeninges was irregular and very slight.

Medulla oblongata. The degree of involvement of the medulla was variable but often moderately severe. There was perivascular round cell infiltration and focal and diffuse gliosis. Both grey and white matter were involved but the damage was usually more severe in the grey. Neuronal degeneration and meningeal infiltration were usually slight.

Cerebellum. The distribution and intensity of the lesions varied considerably, but frequently offered a valuable guide to diagnosis when spinal cord was not

available for examination. Both grey and white matter showed perivascular round cell infiltration and gliosis, but in over 60 per cent. of cases the grey matter showed the greater involvement. Gliosis was usually more focal than diffuse and was more often seen in the molecular than the granular layer. Damage to the Purkinje cells was not a striking feature. Lymphocytic infiltration of the leptomeninges also varied, but was sometimes considerable.

Corpora Quadrigemina. The lesions were essentially similar in type and degree to those seen in the medulla and again tended to be more concentrated in the grey matter. In one case Loyez's method demonstrated rarefaction of myelin in the third cranial nerve.

Cerebrum. The degree of involvement in the cerebrum varied more than in any other part of the central nervous system. Perivascular round cell infiltration of scattered vessels was usually seen coupled with a moderate number of small foci of gliosis. In some cases the process was more severe in the white matter, in others in the grey. Neuronal degeneration was rarely seen and meningeal infiltration was slight or absent.

In some cases the infiltrating cells showed considerable karyorrhexis and the eosinophiles, always present in small numbers in the perivascular cuffs, were then markedly increased in number. This the authors believe to be due to a chemotactic attraction exerted on eosinophiles by degenerating lymphocytes as postulated by Bunting (1932). In all cases polymorphonuclear leucocytes were very rarely seen.

In contrast to swine fever there was little or no vascular damage or infiltration in the choroid plexuses of the brain.

Experimental Investigations

The initial material for these experiments was the brain and spinal cord of the 10-week-old pig from Outbreak V, Group D. It had shown vague signs of ill health on August 5th, 1956, two days after inoculation with crystal violet swine fever vaccine. The pigman administered sulphamezathine, but the pig died on August 7th. The brain and spinal cord were removed on August 8th, 1956, and sent without any preservative to the Weybridge laboratory, where it was given the reference number PD 2572.

Material and methods

Inocula were prepared in beef heart infusion broth buffered to *p*H 7.4. Intracerebral and epidural inoculations were performed under trichloro-ethylene* inhalation anaesthesia. Intracerebral inoculations were made in the mid-line about 1 cm. caudal to the anterior pole of the cerebrum, and epidural inoculations *via* the lumbro-sacral space. The majority of pigs used were drawn from a closed pure-bred Large White herd belonging to the Laboratory. Those used in Experiment II were Large White—Saddleback crossbreds. Those used in Experiments III and IV were the progeny of a Large White gilt produced by the "disease-free" technique. The details and results of the experiments are summarised in Table I. (See overleaf.)

* Trilene. Imperial Chemical Industries Ltd.

Experiment I

Since it was not known at this stage whether the agent was resistant to antibiotics, portions of cerebrum and lumbar cord were emulsified in broth; the emulsion was divided into two parts and to one half were added penicillin and streptomycin to a final dilution of 100 U. per ml. and .001 gm. per ml. respectively. The emulsion was inoculated intracerebrally into 4 one-month-old piglets, PD 2591 and PD 2616 receiving untreated emulsion and PD 2607 and PD 2615 receiving emulsion with antibiotics. The untreated emulsion yielded a considerable growth of coliforms and α-haemolytic streptococci when cultured aerobically for 24 hours on sheep blood agar.

In view of the apparent association between crystal violet vaccination and the onset of the disease in the parent outbreak, PD 2591 and PD 2615 were each inoculated intramuscularly with 5 ml. of crystal violet vaccine on the fourth day of the experiment. By the second day of the experiment. PD 2591 and PD 2616 showed temperatures of 104.2° and 105.8°F. respectively, presumably due to the bacterial contamination of the inoculum. On the second day, therefore, they were each injected intramuscularly with 50,000 U. crystalline penicillin and 250 mg. streptomycin, and on the third day with 250 mg. streptomycin only. On the 11th day, PD 2591 was recumbent and shivering, and if lifted, fell rigidly on its right side. It was killed on the 12th day and was found to have a large cerebral abscess and generalised purulent leptomeningitis which made it difficult to assess any concurrent non-suppurative encephalomyelitis. On the 14th day, PD 2615 showed slight posterior ataxia, which persisted without great change till it was killed on the 25th day. On the 16th day, PD 2607 had difficulty in rising and showed posterior ataxia. By the 17th day all its legs were in a state of flaccid paralysis, but it would still feed if placed near the trough and showed no distress or elevation of temperature. On the morning of the 18th day it was found dead. PD 2616 showed very slight and rather doubtful signs of ataxia on the 16th day but the condition did not progress.

Typical lesions of Talfan disease were found in the central nervous system of PD 2607 and PD 2615.

Experiment Ia.

One ml. of an emulsion of brain and cord from PD 2591 was inoculated intracerebrally into a 3-month-old pig (PD 2651). No significant signs were observed in this pig, which was killed 24 days after inoculation. Traces of a non-suppurative encephalitis were found in the brain of this pig, but the lesions were too slight to be regarded as diagnostic of Talfan disease.

Experiment II

An emulsion was prepared from parts of the cerebrum and cerebellum of PD 2607. To half was added penicillin and streptomycin to a final concentration of 100 U. per ml. and .001 gm. per ml. respectively. The other half was centrifuged at about 2,700 r.p.m. for 5 minutes and the supernatant removed and passed through a Seitz E.K. filter at a pressure of 150 to 200 mm. of mercury.

TABLE I

Experiment number	Number of animal	Age	Inoculation: Inoculum	Route	Amount	Days to symptoms	Days to death	Mode of death	Lesions: Cerebrum	Cerebellum	Corp quad	Medulla	Cord	DRG	Result	Remarks
I	PD 2591	1 m.	Emulsion of PD 2572	i/c	0·6 ml.	11	12	K	±	±	±	±	±	±	±	Cerebral abscess
	PD 2616	1 m	"	"	0·5 "	?16	25	K	—	—	—	—	—	—	—	
	PD 2607	1 m.	Same + antibiotics	"	1 ml.	16	18	D	+	++	++	++	++++	++++	+	
	PD 2615	1 m.	"	"	1 "	14	25	K	+	+++	++	+++	+++	+++	+	
IA	PD 2651	3 m.	Emulsion of brain and cord of PD 2591	i/c	1 ml.	—	24	K	+	±	±	+	±	—	±	
I	PD 2660	8 w.	Emulsion of brain of PD 2607 + antibiotics	i/c & e/d	1 & 1 ml.	?17	22	K	+	±	++	—	—	—	±	
	PD 2667	8 w.		e/d	2 ml.	?14	27	K	—	—	—	—	—	—	—	
	PD 2672	8 w.		i/d		?15	27	K	±	—	—	±	—	—	—	
	PD 2657	8 w.	Seitz EK filtrate of above emulsion	ic/ & e/d	0·75 ; 1 ml.	?20	21	K	—	—	±	—	±	—	—	
	PD 2661	8 w.		i/c	1 ml.	?20	23	K	—	±	—	—	—	—	—	
	PD 2673	8 w.		e/d	1·5 ml.	?20	28	K	—	—	—	—	—	—	—	
	PD 2674	8 w.		i/c	0·75 ml.	?20	29	K	—	—	—	—	—	—	—	
	PD 2675	8 w.	Sediment from filtration	i/n	3 ml.	—	29	K	—	—	—	±	—	—	—	
III	P. 4106	5 d.	Emulsion of brain and cord of PD 2607	i/n	1·25 ml.	—	Not killed									
	PD 2751	5 d.		"	1·25 ml.	—										
	P. 4108	5 d.		"	1·25 ml.	—										
	P. 4109	5 d.		"	1·25 ml.	—										
IV	PD 2750	1 m.	Emulsion of brain and cord of PD 2572 + antibiotics	i/c & e/d	1 & 3 ml.	13	13	K	+	++++	++	++++	+++	++++	+	
	PD 2751	1 m.		"	1 & 3 ml.	15	16	K	+	+++	+++	++++	++++	++++	+	
	P. 4108	1 m.		i n	3 ml.	—	Not killed									
	PD 2759	5½ w.	Seitz EK filtrate of emulsion from PD 2572	i/c	1 "	—	20	K	+	+	—	+	—	—	±	
	PD 2760	5½ w.		i/c & e/d	1 & 0·5 ml.	—	20	K	—	—	—	—	—	—	—	
	PD 2766	5½ w.		i c	1 ml.	—	22	K	—	—	±	—	—	—	—	
	PD 2767	5½ w.		i c	1 "	—	22	K	+	—	+	—	—	—	±	
V	PD 2783	7½ w.	Emulsion of brain and cord of PD 2750	i/c & e/d	1 & 3 ml.	—	19	K	±	—	—	—	—	—	—	
	PD 2797	7½ w.		"	"	—	22	K	+++	++++	+	++	—	—	+	
	PD 2779	7½ w.		"	"	—	16	K	—	—	—	—	—	—	—	
	PD 2805	7½ w.		e/d	4 ml.	—	26	K	—	—	—	—	—	—	—	
	PD 2806	8 w.	Emulsion of brain and cord of PD 2751	i/c	1 "	—	26	K	—	—	—	—	—	—	—	
	PD 2811	8 w.		e/d	3 "	—	27	K	—	—	—	—	—	—	—	
	PD 2814	8 w.		"	3 "	—	28	K	—	—	—	—	—	—	—	
	PD 2817	8 w.		i/c	1 "	—	33	K	—	—	—	—	—	—	—	
VI	PD 2826	4 w.	Collodion filtrate of emulsion of CNS of PD 2572	i/c	1 ml.	12	12	K	+	++++	++	+++	—	—	+	
	PD 2838	4 w.		"	1 "	17	17	K	—	+++	±	+	—	—	+	
	PD 2850	4 w.		"	1 "	?17	23	K	+	—	+	+	—	±	±	
	PD 2851	4 w.		"	1 "	?19	23	K	+	++	++	+++	+	+	+	
	PD 2845	4 w.	Sediment of 2,000 r.p.m. spin of above emulsion + antibiotics	i/c & e/d	1 & 2 ml.	17	22	K	+++	+++	+++	++	++++	++++	+	
	PD 2862	4 w.	Sediment of 10,000 r.p.m. spin of emulsion of PD 2572 + antibiotics	i/c	1 ml.	—	27	K	—	—	—	±	—	—	—	
	P. 4762	4 w.		"	1 "	—	Not killed									
VII	PD 2859	1 m.	Emulsion of CNS of PD 2826 & PD 2838 + antibiotics	i/c & e/d	1 & 1·5 ml.	4	4	D	—	—	—	—	—	—	—	Cerebral abscess
	PD 2876	1 m.		"	1 & 1 ml.	12	12	K	++	+++	+++	++	—	—	+	and suppurative meningitis
VIII	PD 2907	1 m.	Emulsion of brain of PD 2876 + antibiotics	i/c	1 ml.	5	6	K	±	±	±	±	±	±	±	Purulent meningo-encephalitis
	PD 2908	1 m.		"	1 "	6	6	K	±	++	+	++	—	+	±	Doubtful clinical signs
IX	PD 2955	4 w.	Emulsion of cord of PD 2876 + antibiotics	i/c	0·8 ml.	?14	17	K	±	—	±	±	±	—	±	
	PD 2958	4 w.		"	0·7 "	14	18	K	+	+++	++	++++	+	+++	+	Recovering when killed
	PD 2968	4 w.		"	0·8 "	?16	21	K	—	—	—	—	±	±	±	
	PD 2956	4 w.	Emulsion of brain of PD 2876 + antibiotics	"	0·8 "	?13	17	K	—	—	—	±	—	±	±	
	PD 2966	4 w.		"	0·6 "	?14	19	K	—	—	—	—	—	—	—	
	PD 2972	4 w.		"	0·7 "	?18	22	K	—	—	—	—	—	—	—	

K = Killed. D = Died.
— = Negative lesions, signs or result.
± = Doubtful lesions, signs or result.
+ to ++++ = Ascending severity of typical lesions, or positive result.
i/c = Intracerebral. e/d = Epidural. i/n = Intranasal.

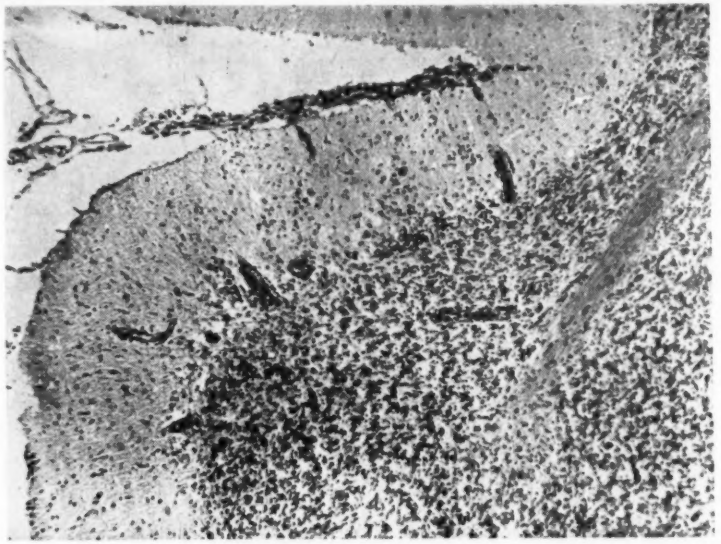

FIG. 1.—PD2572. Natural case (Outbreak V, Group D.) Cerebellar vermis. H. & E. × 80 approx. Showing vascular cuffing and microgliosis in the cortex, and local meningeal infiltration.

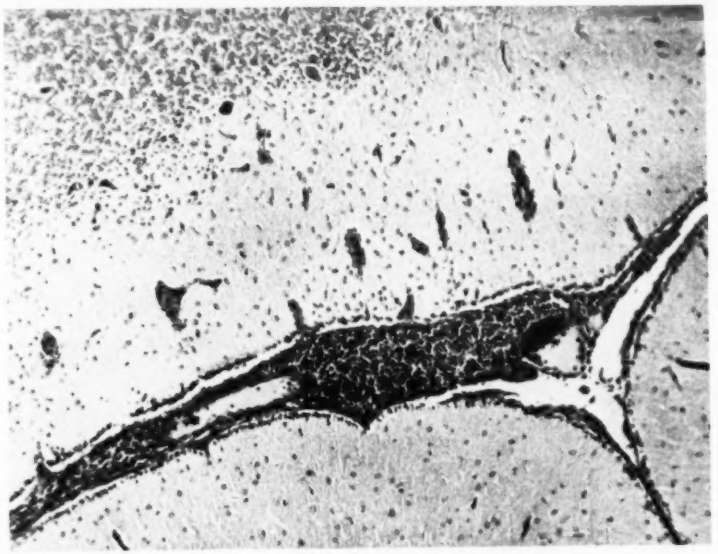

FIG. 2.—PD2845. Experimental case. Cerebellar vermis. H. & E. × 80 approx.

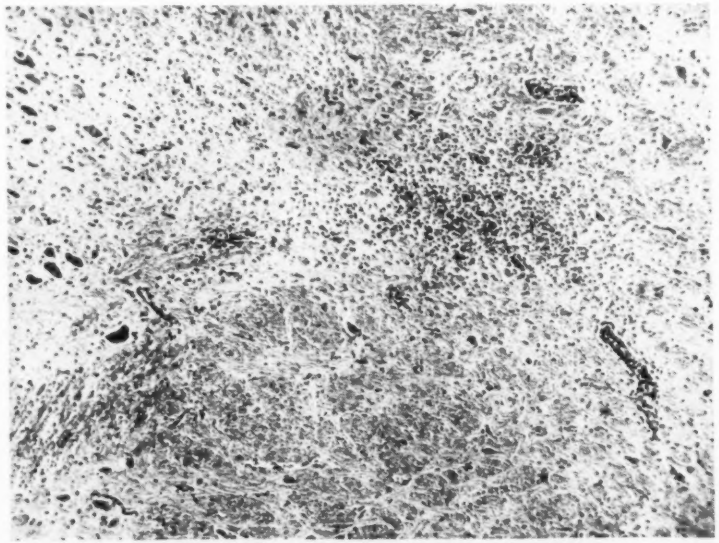

FIG. 3.—PD2572. Medulla oblongata. H. & E. × 80 approx.

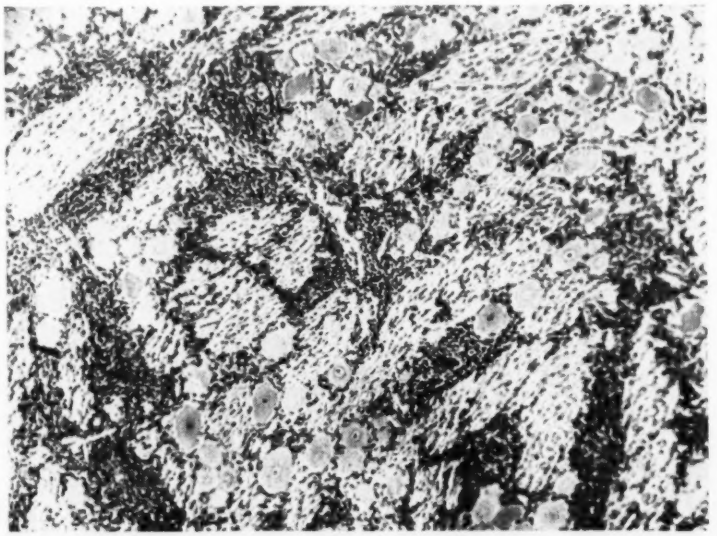

FIG. 4.—PD2572. Dorsal root ganglion from lumbar region. H. & E. × 80 approx. Showing heavy round cell infiltration.

FIG. 5.—PD2572. Ventral horn of dorsal cord. H. & E. × 80 approx. The vascular cuffing and microgliosis is much more obvious in, but not confined to the grey matter.

FIG. 6.—PD2572. Ventral horn of lumbar cord. H. & E. × 80 approx. The lesions are similar to those in the dorsal cord but more marked.

FIG. 7.—PD1311. Natural case: Outbreak H. Anterior cervical cord. Marchi. × 8 approx. This section shows symmetrical demyelination in the dorsal columns.

FIG. 8.—PD2989. Natural case. Lumbar cord. Glee's modification of Marchi. × 8 approx. There is symmetrical demyelination in the dorsal columns and to a lesser extent dorso-laterally and on either side of the ventral median fissure.

The emulsion, filtrate and sediment from centrifugation were inoculated into 8-week-old pigs as shown in the Table. None of these pigs showed definite signs of ill health, and only traces of non-suppurative encephalitis were found in their central nervous systems.

Experiment III

An emulsion of cerebrum, cerebellum and lumbar cord of PD 2607, which had been stored at −15°C. for 14 days, was prepared and 1.25 ml. were inoculated intranasally into 4 out of 6 5-day-old piglets. None of the litter, nor their dam, showed any signs of ill health and they were not killed. The dam had previously been inoculated with attenuated (lapinised) swine fever virus and there is, therefore, some reason to believe that the piglets may have been passively immune to swine fever.

Experiment IV

An emulsion of cerebrum, cerebellum and lumbar cord of PD 2572 which had been stored at −15°C. for 26 days, was prepared. To one half was added penicillin and streptomycin to a final concentration of 1,000 U. per ml. and .01 gm. per ml. respectively. The other half was centrifuged at 2,700 r.p.m. for 5 minutes and the supernatant passed through a Seitz E.K. filter under a pressure of 175 mm. of mercury.

(a) The emulsion and sediment from centrifugation were inoculated as shown in the Table, into 3 piglets of the same litter used in Experiment III. Signs of slight ataxia appeared in PD 2750 on the 13th day and in PD 2751 on the 15th day. As it was suspected that this disease might be the same as that described by Bendixen (1954) as enzootic paresis and by Bendixen and Sjolte (1955) as *Poliomyelitis suum*, these pigs were killed early in the clinical disease, lest the agent should be lost. None of the other pigs of the litter showed signs of ill health and therefore none was killed.

(b) The filtrate was inoculated into 4 5½-week-old pigs as shown in the Table. None of these pigs showed signs of ill health and only slight lesions of non-suppurative encephalitis could be detected in their central nervous systems.

Experiment V

Two emulsions were prepared, from the cerebra, cerebella and spinal cords of PD 2750 and PD 2751 respectively, and were inoculated into 7½- and 8-week-old pigs as shown in the Table. None of these pigs showed any significant signs, but in the brain of PD 2797 it was possible to demonstrate typical lesions of Talfan disease. This is the only case in which we have been able to reproduce the condition in pigs as old as 7 weeks at the time of inoculation.

Experiment VI

An emulsion was prepared from the cerebrum and cerebellum of PD 2572 which had by now been stored at −15°C. for 94 days. This was centrifuged at 2,000 r.p.m. for 15 minutes; the supernatant was removed and centrifuged at 10,000 r.p.m. for 50 minutes and the sediment was resuspended in broth. The supernatant from the second centrifugation was passed through a collodion membrane of an average pore-diameter of 590 mμ under a pressure of 740 mm. of water in about two minutes. Culture of this filtrate aerobically on sheep blood agar gave no bacterial growth. The sediment from the second centrifugation was also resuspended in broth, and penicillin and streptomycin were added to the resuspended sediments to a final concentration of 1,000 U. per ml. and .001 gm. per ml. respectively. These preparations were inoculated into 4-week-old pigs as shown in the Table.

Typical lesions of Talfan disease were demonstrable in the central nervous systems of 3 out of the 4 pigs receiving filtrate and 1 out of the 3 pigs receiving sediment, but typical signs were only seen in PD 2826, PD 2838 and PD 2845.

Experiment VII

An emulsion was prepared from the cerebra, cerebella and lumbar cords of PD 2826 and PD 2838 and inoculated into two 4-week-old pigs as shown in the Table. PD 2859 died on the fourth day, and showed a large cerebral abscess and widespread purulent leptomeningitis. PD 2876, however, showed typical ataxia on the 12th day, was killed, and showed typical lesions of Talfan disease.

Experiment VIII

An emulsion was prepared from the cerebrum and cerebellum of PD 2876 with penicillin and streptomycin added to a final concentration of 1,000 U. per ml. and .001 gm. per ml. respectively; this was inoculated intracerebrally into two 1-month-old pigs. On the fifth day of experiment PD 2907 showed signs of distress, rigid recumbency and opisthotonos. On the sixth day, PD 2908 developed similar signs and both were killed. PD 2907 showed lesions of purulent meningitis tending to confuse lesions of non-suppurative encephalitis. PD 2908 showed mild but typical lesions of Talfan disease. In view of the atypical clinical signs and incubation period, it was concluded that there had possibly been an error in the technique of inoculation and it was decided to repeat this passage.

Experiment IX

Two emulsions were prepared, one from the cerebrum and cerebellum and one from the spinal cord, of PD 2876, penicillin and streptomycin were added to both to give a final concentration of 1,000 U. per ml. and .001 gm. per ml. respectively. The emulsions were inoculated into 6 4-week-old pigs as shown in the Table. On the 14th day of experiment PD 2958 showed marked inco-ordination, falling frequently and having difficulty regaining its feet. On the 15th day, however, the pig showed only slight inco-ordination, which became progressively less until it was killed on the 18th day. The other pigs in the experiment showed only very slight and indefinite signs of posterior inco-ordination. Typical lesions of Talfan disease were found in the central nervous system of PD 2958.

It will be seen that experiments VI to IX constitute 3 serial passages of the infectious agent.

The Experimental Disease

Clinical signs of the disease were seen only in pigs inoculated by intracerebral and/or epidural (lumbo-sacral) routes. Since intranasal inoculation failed to produce clinically obvious signs, no pigs inoculated by that route were killed and it is not known whether mild lesions were in fact present. However, since pigs which survived Experiment III were successfully infected in Experiment IV 30 days later, this seems unlikely.

Typical signs and lesions were reproduced in 10 out of 26 pigs which were not more than one month old at the time of inoculation. In pigs older than one month, signs were seldom seen and, although some evidence of nonsuppurative encephalitis was present in the central nervous system of 3 out of 20 only one showed lesions sufficiently well developed to be identified with certainty as a positive transmission (PD 2797).

The severity of clinical signs produced varied considerably. Some cases showed only a mild posterior weakness and inco-ordination. In the majority, however, the typical course of the disease was as follows:—

After an incubation period of 12+ days, the animal would show a slight posterior ataxia. This usually progressed rapidly and within 8 hours there was marked inco-ordination and paresis. By 24 hours after the onset of signs, the animal was usually recumbent with flaccid paralysis of all 4 limbs. There were no obvious signs of distress and, if assisted to the trough, it would continue to feed. The temperature at this stage was usually within normal limits. In less severe cases several days of severe inco-ordination might intervene between the onset of signs and the stage of paralysis. In the final phase the pig would become quietly depressed, lose interest in food and was then usually killed. The period between paralysis and death was variable, PD 2607 died within 2 days of the onset of signs, but other pigs survived for as long as 11 days and were then killed.

An intermittent dark diarrhoea was frequently noted during the incubation period, but since the pigs had often only been weaned on to a propriety sow milk substitute a few days before the commencement of experiment, it is not clear whether or not this was caused by the experimental agent.

The lesions found in the central nervous system compare closely in type and distribution with those of the natural disease, but are generally rather more severe. The lesions of the cerebellum are particularly striking. Throughout the rest of the central nervous system involvement of the meninges is slight, patchy and inconstant, but over the cerebellum there is a very severe non-suppurative leptomeningitis, which is accompanied by severe focal and diffuse gliosis and perivascular round cell infiltration, almost invariably more severe in the grey matter than in the white. In some of the pigs inoculated intracerebrally which were killed very soon after the onset of clinical signs, the typical lesions of the spinal cord and dorsal root ganglion were not present, but it is considered that the cerebellar lesions, when well developed, are sufficiently characteristic to justify a claim for positive transmission. In Experiment IX, 4 pigs were inoculated with spinal cord which had not shown lesions, yet a transmission was established.

No consistent abnormalities could be detected in liver, lung, myocardium, testes, small intestine, coeliac ganglion, pancreas, pituitary, thyroid, adrenal, spleen or lymph node from the experimental pigs. Small foci of lymphoid infiltration were frequently seen in the renal medulla but, since these were present in both positive and negative transmissions, and were also observed in pigs from the same source used on other experiments, it seems unlikely that they are associated with Talfan disease.

Discussion

The evidence presented suggests that there is, in Great Britain, a nonsuppurative encephalomyelitis, usually affecting pigs of the suckling and immediately post-weaning age, though histologically indistinguishable lesions have been seen in pigs as old as 8 months. Typically, the morbidity and mortality is low and the picture is one of a severely ataxic or flaccidly paralysed pig without significant elevation of temperature or loss of appetite. Recovery occurs in a proportion of animals which are hand fed. "Convulsions" have been described in some outbreaks, but these have often not been seen by a veterinary surgeon and may merely represent the struggles of the animal to move about. Cerebral signs are certainly not a feature of the experimental disease.

The disease appears to be caused by a transmissible agent which resists penicillin and streptomycin at a concentration of 1,000 U. per ml. and .001 gm. per ml. respectively and freezing at $-15°$ C. for at least 94 days, and is capable of passing a collodion membrane of 590 mμ average pore diameter. Evidence as to whether the agent will pass a Seitz E.K. filter is inconclusive at this stage. The minimum experimental incubation period of the disease is 12 days, and the agent can be passaged serially in pigs up to one month old by intracerebral inoculation of brain and spinal cord. There is no evidence that inoculation with crystal violet swine fever vaccine has any influence on the course of the disease.

One must consider whether the disease is identical with any of the known non-suppurative encephalomyelitides of pigs.

Swine Fever. The incubation period of swine fever, after intracerebral inoculation is 3 to 4 days and results in a mortality of 100 per cent. The lesions of the central nervous system are usually most severe in the mid and hind brain. Severe involvement of the spinal cord and of the grey matter of the cerebellum is rare and the white matter of the cerebellum is usually more severely affected than the grey. There is also a considerable degree of degeneration in the endothelium of the affected vessels, and the choroid plexuses of the brain are involved as well as nervous tissue proper. The clinical and *post-mortem* picture of naturally occurring swine fever is obviously very different from that of Talfan disease.

Louping Ill. Louping ill has rarely been reported as a naturally occurring disease of pigs. Pool, Brownlee and Wilson (1930) infected pigs intracerebrally and found a temperature reaction after 1 to 2 days, and on the fifth day, hyperaesthesia tremors of the head and limbs and paralysis. They also transmitted the disease by subcutaneous inoculation. Brownlee and Wilson (1932) emphasise the intense infiltration of the meninges of the cord, which is never a feature of Talfan disease.

On histopathological evidence alone it might be possible to confuse the two diseases, but it is the authors' impression that the cerebellar damage and glial reaction is much more specifically centred on the Purkinje cells in experimental louping ill than in Talfan disease. In none of the outbreaks reported was there any evidence that the pigs had had contact with ticks. Sera from pigs Nos. PD 2591, PD 2615 and PD 2616 were examined by Dr. J. T. Stamp, who reported " no louping-ill antibodies present."

Aujeszky's Disease. Severe and fatal cases of naturally occurring Aujeszky's disease are virtually confined to young pigs and clinical signs attributable to involvement of the cerebrum usually predominate. Microscopically the more anterior parts of the central nervous system are usually found to be the most severely affected, neuronal damage is heavy and widespread and characteristic intranuclear inclusions are present in a good proportion of the most seriously affected neurones and some glial cells (Done, 1954).

Intracerebral inoculation of Aujeszky's virus is constantly fatal to pigs, usually in from 3½ to 8 days (Hurst, 1933; Shahan, Knudsen, Seibold & Dale, 1947) and again the cerebrum is the most severely affected part of the brain. After non-fatal infections with Aujeszky's virus neutralising antibodies are present in the serum of pigs in about 17 days (Done, 1955) but no such antibodies could be demonstrated in the sera of pigs PD 2591, 2615, and 2616, 12, 25 and 25 days respectively after exposure to the agent of Talfan disease.

Teschen Disease. The differential diagnosis of Teschen disease has been discussed at some length by Bendixen and Sjolte (1955) and it is not proposed to repeat the discussion in full. However, the work of Horstmann, Manuelidis and Sprinz (1951) and Horstmann (1952) gives a clear picture of the common findings. By intracerebral inoculation 15 out of 15 pigs were successfully infected with an incubation period ranging from 4 to 10 days; by intranasal inoculation 8 out of 12 with an incubation period of 7 to 17 days, and by oral infection 11 out of 15 with an incubation period of 11 to 15 days. All these pigs showed a temperature rise of at least 2° C. Histologically, lesions were practically confined to the grey matter, while the lesions of Talfan disease are to be found in both grey and white matter, though usually more severe in the grey.

Japanese B Encephalitis. The authors have had difficulty in finding a detailed histopathological account of this disease in the pig and little pathological material is available to them. However, Tsubaki, Masu and Shimada (1949) emphasise amongst other features of the encephalitis degeneration of the Purkinje cells, atrophic degeneration of ganglion cells and haemorrhagic lesions. None of these lesions are conspicuous features of Talfan disease. Ochi (1953) indicates that the nervous manifestations of the disease in the young pigs are usually accompanied by abortions or stillbirths after the expected time of parturition in 50 to 70 per cent. of the pregnant females in the herd.

Unidentified myelitis. Doyle (1937) described a posterior paralysis of swine which he believed to be infectious. His description of the histopathology of the disease is, however, not sufficiently detailed to act as a basis for a discussion on differential diagnosis

Poliomyelitis suum. The clinical and pathological description of this disease given by Bendixen and Sjolte (1955) compares closely with Talfan disease. In Denmark, however, the most common age group affected by the naturally occurring disease was 12 to 16 weeks, while a lower incidence occurred in pigs 4 to 8 weeks and over 16 weeks of age. In England and Wales the disease is most often seen in pigs below 10 weeks of age. Experimentally, *Poliomyelitis suum* could be reproduced by intracerebral or intranasal inoculation, with an incubation period of 14 to 25 days, averaging 18.1 days.

Of the non-suppurative encephalomyelitides considered, it appears that *Poliomyelitis suum* is the only condition closely resembling Talfan disease and, in view of the differences outlined above, the relationship is still uncertain. Christensen (1956) in a private communication indicated that he had been unable to establish a satisfactory serological test for *Poliomyelitis suum*; it therefore seems impossible at this stage further to clarify the position.

Even if the two diseases are identical, *Poliomyelitis suum* seems to be an undesirable name, since that and other very similar titles have already been used for Teschen disease and, from a histopathological point of view, the name could be applied with equal force to several other encephalitides of swine. For these reasons the authors, while mindful of the philosophical precept that *" essentia non sunt multiplicanda praeter necessitatem "* (Occam), prefer to call the condition, seen in England and Wales, Talfan disease.

Summary

1. The clinical and pathological features of a polioencephalomyelitis of pigs occurring naturally in Great Britain and hitherto unrecorded, are summarised.

2. Experiments demonstrating that the aetiological agent is probably a virus, are described.

3. The relationship of the agent to other viral encephalomyelitides of swine is discussed.

Acknowledgments.—To the V.I.O.s. and A.V.I.O.s. indicated in the text and to Messrs. P. M. Bennett, B.SC., M.R.C.V.S., and F. C. Baker, B.SC., M.R.C.V.S., all of whom supplied material and information; to Dr. J. T. Stamp (Moredun Institute) and Mr. W. Mansi, M.V.SC. (CAIRO), DIP. BACT. (WEYBRIDGE), who both examined sera; to Mr. R. A. Huck, B.SC., M.R.C.V.S. (WEYBRIDGE), who performed the col-

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Observations on Copper Deficiency in Cattle in East Anglia

BY

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Part II

Response to Treatment

THE remaining experiments were carried out during 1953 and 1954 amongst cattle on another large reclaimed area of fenland in Norfolk. This fen was partly reclaimed during the war and the area of reclamation was subsequently extended. No cattle were kept on the reclaimed land until the autumn of 1952, when some 2,000 Irish stores, varying in age from 15 to 18 months, were purchased and brought on to new leys (mainly cocksfoot) on the fen. The stores ran out during the winter of 1952-3, being fed on beet tops, fodder beet, and hay. The intention was to sell the cattle fat off the pastures during the summer and autumn of 1953 but in the summer many of the cattle started to scour and lose condition and failed to make the weight for grading. As a result, some 600 to 700 were kept on for yard feeding during the winter. In July, 1953, blood samples from 8 affected cattle showed average blood copper levels of 0.015 mg. per 100 ml. of blood, as compared with the average normal of around 0.09 to 0.10 mg. per 100 ml. The management remained unconvinced that copper deficiency was solely responsible for the lack of thriving in the stock and in order to produce evidence which might be fully acceptable, a small experiment was carried out which was dramatic in its effect and fully achieved the object for which it was intended.

Treatment by Intravenous Injection (Experiment 4)

Experimental Details

Twelve cattle were selected at random from a group of affected stores and weighed individually. Six of the beasts were each given an intravenous injection of approximately 350 mg. of copper as $CuSo_4$ in September, 1953, and another similar dose in April, 1954. The other 6 beasts were kept as untreated controls. The 12 cattle were maintained at pasture until sent for slaughter in the autumn of 1954 but received supplementary feeding during the winter of 1953-4.

Experimental Results

The cattle were weighed at intervals from September, 1953, until they were slaughtered in October, 1954. The results are shown in Fig. 5. The 6 treated cows made a dramatic response to treatment, making an average live-weight gain of 135 lb. in the 70 days following treatment as compared with an average increase of 23 lb. in the 6 controls. Photographs of typical animals before and subsequent to treatment are shown in Figures 6 and 7. During the winter the cattle in both groups lost weight but started to improve again with the spring flush of pasture. In October, 1954, approximately 13 months after the first treatment with $CuSo_4$, the cattle in the treated group showed an average live-weight increase over the experimental period of 453 lb. as compared with 362 lb. in the controls.

The cattle were slaughtered at the beginning of November, 1954, the grading weights being as shown in the table overleaf.

Blood samples were collected at intervals for analysis from cattle in both the treated and control groups. The blood copper values are given in Fig. 8. The levels show that the single intravenous injection given in September was sufficient to raise the blood

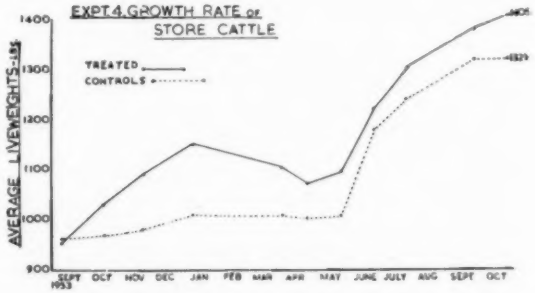

FIG. 5.

A Transmissible Polio-encephalomyelitis of Pigs (Talfan Disease).—*Concluded.*

lodion membrane filtration, and to Miss M. D. Windsor, M.R.C.V.S. (WEYBRIDGE), who assisted with anaesthesia and *post-mortem* examinations.

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EXPERIMENT 4.

Treatment of 2-year-old copper-deficient store cattle by intravenous injection.

FIG. 6.—Typical case before treatment.

FIG. 7.—Representative animal of group after treatment.

Treated cattle		Untreated cattle	
Number	Killing out weight/ lb.	Number	Killing out weight/ lb.
1	767	1	738
2	836	2	722
3	866	3	792
4	824	4	749
5	705	5	676
6	800	6	745
Averages :	833		737
Killing out percentages :	59·3		55·7

copper levels to within the normal range. The levels were still normal in 3 out of 5 cattle 7 months later, that is, in April, 1954. A second injection at the end of April restored the blood levels in the remainder and normal levels were maintained until the termination of the experiment. Throughout the experimental period the treated cattle invariably showed higher haemoglobin levels than the controls (see Fig. 8).

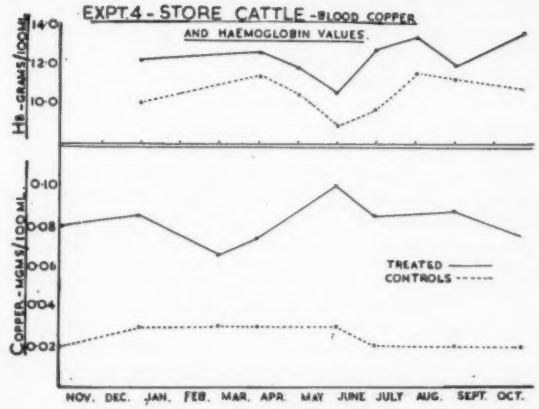

FIG. 8.

Analyses of the liver samples collected from each animal at the time of slaughter showed an average liver content of 30.65 p.p.m. in the treated cattle as compared with an average level of 6.05 p.p.m. in the untreated controls.

Treatment by Feeding Anti-teart Cake (Experiment 5)

In addition to feeding off Irish store cattle, some of the new leys in this reclaimed area were used as summer grazing for young dairy stock from farms in the same ownership in other parts of Norfolk. In March, 1953, a large group of young Friesian and cross-bred Friesian cattle arrived on the fen and were put on the young leys. These cattle varied in age from 9 to 15 months. Despite the lush appearance of the pastures the young stock failed to thrive and by September were showing characteristic symptoms of copper deficiency, the main features being a tucked-up appearance of the abdomen, pronounced scouring, greying of the coat colour and, in some cases, loss of hair around the eyes. These features are well illustrated in Figs. 9 and 10. Because of the large number of animals available for trial, an experiment involving the administration of copper by the intravenous route was not considered to be practicable and a trial was planned involving the use of anti-teart cake (a specially compounded cake containing $CuSo_4$).

Experimental Details

Eighty cattle were taken for experiment and divided at random into 2 equal groups of 40. The cattle were weighed on October 12th and the groups put on separate pastures of similar size and botanical composition. They were subsequently trough fed daily, 1 group receiving ration A and the other group ration B.

Ration A

60 per cent. oats
20 per cent. wheat
20 per cent. anti-teart cake

Ration B

60 per cent. oats
20 per cent. wheat
20 per cent. milk nuts

These rations varied in composition only to the extent that the anti-teart cake contained copper whereas the milk nuts in ration B did not. Each group received 112 lb. of the appropriate ration daily until December 7th, when, partly for humanitarian reasons, the cattle which had previously received ration B were changed to ration A.

EXPERIMENT 5.

FIG. 9.—Copper deficiency in cross-bred Friesian stores.

FIG. 10.
Showing symptoms of debility, fading of coat colour, loss of hair around the eyes and scouring. (Autumn)

Experimental Results

The cattle were examined and again weighed on November 25th, that is, after the supplementary food had been supplied for 44 days. By this time the group receiving ration A were much improved clinically and had ceased scouring. The average weight increase of cattle in this group from October 12th to November 25th was 133 lb. or approximately 3 lb. per head per day. The cattle receiving ration B had deteriorated in condition and during the same period lost an average of 35 lb. in weight (see Fig. 11).

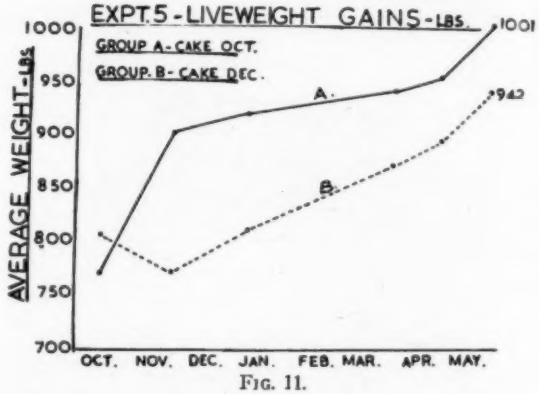

FIG. 11.

Six cattle were bled from each group for blood copper estimation on December 1st. Those on ration A showed average values of 0.08 mg. per 100 ml. of blood, whereas those on ration B showed average values of 0.017 mg. per 100 ml.

The cattle in both groups were given a daily ration of hay and fodder beet as from December 1st and because it was felt that some of the cattle on ration B might die from debility this group was also transferred to ration A on December 7th. Despite the change to a ration which contained available copper, however, the second group did not make the same dramatic response that had occurred in the cattle which received ration A in October. This was probably due to the fact that the cattle were mainly dependent in December on supplementary feeding, the herbage no longer being of a high nutritive quality. Blood samples were take from cattle in both groups in January, March, April, and May, and at all tests the blood copper values were within the normal range. The feeding of anti-teart cake ceased on March 27th, 1954.

The cattle in both groups were weighed for the last time on May 26th, when it was found that the cattle which received ration A as from October 12th had made an average weight increase of 231 lb. in 226 days, as compared with an average weight increase of 137 lb. in the same period for the cattle which received ration A from December onwards.

In June, 1954, some of the heifers from the experimental group of 80 were selected for breeding and the remainder sold as stores in the open market.

The 53 stores sold at market averaged over £57 per head which worked out at just over £6 per live cwt. The cost of feeding anti-teart cake to the cattle from October 12th to March 27th (168 days) was approximately 30s. per head.

The Mineral Content of Fen Pastures and its Relationship to the Development of Copper Deficiency in Grazing Cattle

In some areas, copper deficiency may be due to a simple and uncomplicated shortage of copper in the herbage, while in other areas where the copper content of the herbage is adequate to meet the nutritional

requirements of stock, the deficiency may be induced by an excess of molybdenum or some other dietary factor which prevents the utilisation of copper. Where excess of molybdenum is involved there are accompanying symptoms of molybdenum intoxication (Cunningham, 1955).

Cunningham (1950) gave the copper content of normal pastures in New Zealand as 11 parts per million (p.p.m.) on a dry-matter basis and the molybdenum value as 1 to 2 p.p.m. These figures are similar to those reported in England by Allcroft (Allcroft & Parker, 1949) who found that the copper and molybdenum contents of laboratory pastures averaged 11.4 and 1.3 p.p.m. respectively.

In those parts of New Zealand and Australia where symptoms in cattle are ascribed to uncomplicated copper deficiency, the copper content of the pastures averages 3 p.p.m., this level being insufficient to meet the physiological requirements of the grazing animal. The symptoms reported for this type of deficiency are unthriftiness, seasonal anaemia, and in the female, disturbance of oestral cycle, sometimes causing temporary infertility. If the deficiency is severe, for example, on pastures with 1 to 2 p.p.m. copper, sudden deaths occur. But copper deficiency in cattle, as evidenced by low blood and tissue levels, also occurs on pastures which contain adequate copper. Cunningham (1955) considers that pastures with a content higher than 5 p.p.m. will supply enough copper, and that where diseases of cattle occur in which there is a deficiency of copper within the animal but not necessarily in the food, such diseases are associated with an accompanying dietary excess of molybdenum or some other dietary factor which interferes with copper assimilation. Cunningham calls this kind of copper deficiency " induced " or " complicated." The main feature which clinically distinguishes induced from simple copper deficiency is that scouring commonly occurs in the former but is rare in the latter.

Cunningham and his colleagues have shown quite clearly that molybdenum is important in " peat scours " in New Zealand. This condition occurs on pastures with an average copper content of 7 p.p.m. Analysis of pastures in 2 areas in which the disease occurred showed a seasonal variation in the molybdenum content, but no variation was reported in the copper levels. The monthly molybdenum levels were as follows:—

Area	Molybdenum in pasture—parts per million in dry grass											
	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Kerepehi ...	—	3·2	2·5	2·5	4·1	7·0	6·2	7·4	6·2	4·8	2·8	2·9
Gordonton ...	—	7·5	7·6	7·5	10·2	15·0	16·5	14·3	16·5	11·2	7·9	8·8

The first indication which led to the establishment of a relationship between molybdenum and copper came from the effectiveness of daily doses of copper in controlling the profuse scouring of " teart," a condition caused by high concentrations of molybdenum in pastures in parts of Somerset (Ferguson, Lewis & Watson, 1943). It has since been shown experimentally that dietary molybdenum will affect the storage of copper in cattle (Dick & Bull, 1945; Cunningham, 1950; Davis, 1950; Allcroft, 1952). When dietary copper is around normal, an increase in molybdenum intake reduces the copper already stored in the liver and the amount of copper deposited there from the food. Cunningham (1954) states that molybdenum levels below 3 p.p.m. have no effect; a level of 3 to 10 p.p.m. is harmful if the copper intake is below normal, and above 10 p.p.m. is harmful even if the copper intake is normal.

The investigations carried out in Great Britain since copper deficiency in cattle was first reported in 1940 have failed to provide acceptable evidence of a simple deficiency or an induced deficiency due to an excess of molybdenum. Pastures have generally been regarded as having a normal copper content and Green (1949) did not regard molybdenum as having any part in induced copper deficiency in Great Britain. It is postulated that there must be some other dietary factor which interferes with the assimilation of copper. There is, however, a lack of detailed information in the literature on the results of pasture analyses. This aspect of the problem appeared to warrant more intensive study, and for this reason pasture samples were collected from 4 fen pastures for analysis every 2 or 3 weeks throughout 1 year from January to December inclusive. The samples were analysed for copper and molybdenum.

The analytical results are presented in 2 ways. In Fig. 12 the levels for the individual fields are shown in the form of graphs, while in Table VIII they are shown under 3 periods of the year, *viz.* January to April inclusive, May to September inclusive, and October to December inclusive. The copper levels followed a consistent pattern in each of the 4 fields, being lower during the grazing season from May to September than they were during the rest of the year, but even during this period the content was adequate to supply the nutritional requirements for copper of the grazing animal. The molybdenum levels of 2 pastures (122 and 131) were at all times of the year substantially higher than the others (White Fen and 1248). Furthermore, the molybdenum content of these pastures rose during the grazing season, when the copper levels were falling, and reached levels equal to and at times greater than the copper content. There was, therefore, during the period from May to September an inverse relationship between the copper and molybdenum contents of these fields.

The average molybdenum levels of fields 122 and 131 during the grazing season exceeded the level of 10 p.p.m. which Cunningham holds to be harmful even when the copper content is normal. It might therefore be argued on this evidence that the analytical

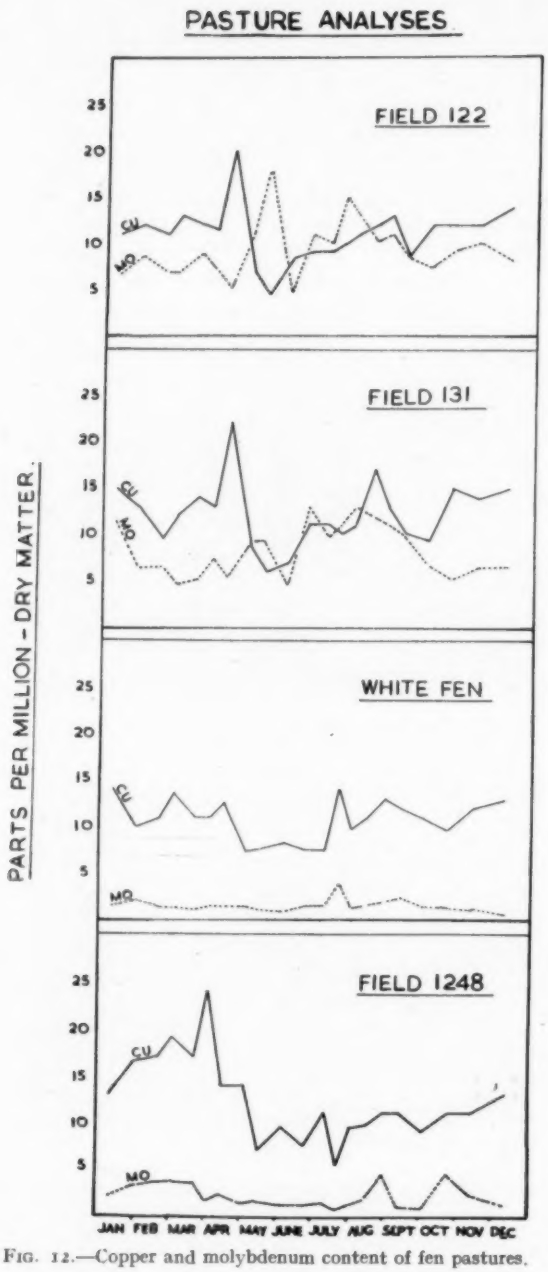

FIG. 12.—Copper and molybdenum content of fen pastures.

results are in keeping with an induced copper deficiency due to an excess of molybdenum and, indeed, the symptoms of copper deficiency as seen on the fens are similar to those described by Cunningham for "peat scours." However, the molybdenum levels of the other fields were of an order which have been found to have no effect on copper assimilation.

Although cattle on the fens become deficient in copper, it is quite clear from these analytical results that it is not due to a deficiency of copper in the herbage, although it is now established that there is some seasonal variation in the copper content of the pastures. The deficiency is, therefore, induced in type. It is logical to suppose that the factor which interferes with copper assimilation is common to the whole area and that if this factor was excess molybdenum it might have been expected to show itself in the analytical results from each of the 4 fields. In the event, this evidence was not obtained and the case against molybdenum must be regarded as not proven

Assessment of Results

Although these experiments were all carried out in East Anglia, the results have a wider interest, as copper deficiency has been confirmed in cattle in many parts of Great Britain during the last 10 years.

Those who are concerned solely with research might criticise certain features of the planning and conduct of some of the studies, such as Experiments 1, 3, and 5, on the grounds that the groups did not contain equal numbers of animals or that some of the experiments were altered or terminated prematurely. It must be remembered, however, that studies of this kind carried out by the staffs of Investigation Centres are necessarily of an *ad hoc* nature and, what is perhaps even more important, the officers must strike a balance between the interests of the farmer and a desire to add to available knowledge on a given disease. In the event, it is usually possible to arrive at a compromise arrangement which enables the farmer to greatly reduce his losses while at the same time leaving room to gain further information about a disease when it would be of value to do so. This reasoned approach explains the variations in the size of treated and control groups respectively in Experiments 1 and 3 and also explains why the control group in Experiment 5 was changed to a ration containing copper after an experimental period of only 2 months. Despite the limitations which this approach must necessarily impose on the study of problems of this kind, some useful information has been obtained on the general problem of copper deficiency in cattle and its control.

The Disease in Calves

Copper deficiency is a problem which is mainly confined to the grazing season. The data presented in Table VIII show that during this period of the year the copper content of the herbage shows a seasonal fall. Cattle grazing fen pastures have extremely low copper levels, liver values falling to less than 10 p.p.m. and blood values to 0.01 mg. per 100 ml., as compared with normal values of 200 p.p.m. and 0.1 mg. per 100 ml. The figures given in Tables I and IV show that if cows are copper deficient during pregnancy then their calves are deficient at birth, as evidenced by low liver and blood values. The calves are unable to make good this deficiency from the milk. The results obtained in Experiments 1 and 2 show that such deficient calves develop signs of deficiency when 2 to 3 months of age. If, however, the copper status of the cows is

TABLE VIII

VALUES FOR PASTURE SAMPLES—SHOWING DIFFERENCES IN COPPER AND MOLYBDENUM LEVELS ACCORDING TO SEASONS

Field	Number of samples	Period	Copper*		Molybdenum†	
			Average value	Range	Average value	Range
122	7	January to April inclusive	13·0	11·0 to 12·0	7·4	5·1 to 9·0
	10	May to September (main grazing season)	9·25	4·4 „ 13·0	11·0	4·7 „ 18·0
	4	October to December inclusive	12·5	12·0 „ 14·0	8·7	7·5 „ 10·0
131	7	January to April inclusive	14·1	9·6 to 22·0	6·7	4·8 to 11·5
	10	May to September inclusive	10·5	6·1 „ 17·0	10·25	4·7 „ 13·0
	4	October to December inclusive	13·3	9·4 „ 15·0	6·25	5·2 „ 6·8
White Fen	7	January to April inclusive	11·7	10·0 to 14·0	1·6	1·3 to 2·2
	10	May to September inclusive	9·87	7·5 „ 14·0	1·88	1·0 „ 4·0
	4	October to December inclusive	11·4	9·7 „ 13·0	1·3	0·8 „ 1·6
1248	7	January to April inclusive	17·2	13·0 to 24·0	2·9	1·7 to 3·7
	10	May to September inclusive	9·58	5·4 „ 14·0	1·67	0·9 „ 4·4
	4	October to December inclusive	11·0	9·0 „ 13·0	2·2	0·9 „ 4·5

* Normal average content 11 p.p.m.—dry matter. † Normal average content 2 p.p.m.—dry matter.

maintained during pregnancy, then calves are born with adequate liver reserves and blood levels and thrive in a normal way. The data supporting these conclusions are presented in Fig. 4 and Tables III and IV. Copper deficiency in calves can also be prevented by providing copper to the cows and calves during the suckling period, as shown by the results of clinical observations in Experiment 2.

Deficiency in Older Stock

It seems quite clear from the results obtained in Experiment 3 that it is essential to maintain the copper status of cattle above the deficiency level throughout the growth period if normal growth rate is to be maintained. Over an 18-month observation period, a group of cattle in which the individuals were copper deficient throughout made an average daily weight gain of 0.69 lb.; a second group deficient for part of the time, an average of 0.89 lb., and a third group in which the copper status was maintained around normal throughout, 1.09 lb. The total difference in weight gain in 18 months between the first and third of these groups was 203.2 lb.

Deficiency in the Adult

When copper deficiency is having a marked clinical effect on young and growing cattle, there is a tendency to ignore the less-apparent but no less important effects of deficiency in the adult cow. The clinical observations which formed part of Experiment 1 showed that towards the latter part of the grazing season there was an appreciable decline in the physical condition of deficient cows, shown by general unthriftiness and fading of the coat colour. The extremely low levels to which the copper content of the blood and livers of adult cattle can fall is shown by the figures included in Tables I and IV. The haemoglobin levels also fall during the grazing season but not to a level likely to give rise to anaemia. According to Holman (1955) pathological anaemia is unlikely to occur until the haemoglobin level falls well below 8 grammes per 100 ml. In the deficient cows in Experiment 1 the lowest average level, which was reached in mid-September, was 8.34 grammes per 100 ml. of blood. In a later experiment involving 2- to 3-year-old stores (Experiment 4), deficient cattle consistently showed lower Hb. levels than normal cattle (Fig. 8).

Methods of Prevention

The prevention of copper deficiency is essentially a problem of maintaining liver and blood levels at or around normal. Cattle invariably develop symptoms of deficiency during the grazing season, usually after they have been at pasture a few months. One way of preventing the disease is to provide the cattle with copper during this period. A method commonly used in Australasia is to spray the pastures each spring with $CuSo_4$. A rate of 5 lb. per acre is generally employed. That this method can also be effective in this country is shown in the results obtained in Experiment 2. Care needs to be exercised if sheep are also kept on the pastures in view of the susceptibility of this species to copper poisoning.

If spraying is impracticable, other methods may be considered, *e.g.* oral dosing at 7- to 14-day intervals, or intravenous injections. The former method is laborious and time consuming but nevertheless effective, as is shown by the results of Experiment 1. The effects of a single intravenous injection, as employed in Experiment 4, appear to last for 6 to 7 months and this may be the method of choice with small numbers of animals. But copper deficiency can also be prevented by building up the liver and blood levels of cattle during the winter preceding the grazing season. This method was used in Experiments 1 and 3. If this is done, there is no need to supply available copper to the stock during the grazing season. The store of copper present in the liver when the cattle go out to grass is sufficient to maintain them until the autumn. This build-up during the winter can be achieved by oral dosing, intravenous injection, or,

more conveniently, by incorporating in the diet ½ lb. per head each day of so-called " anti-teart " cake, which contains the equivalent of 2 grammes of $CuSo_4$ per lb.

The intramuscular injection of copper glycinate has been used recently in Australasia as a means of supplying available copper and in areas where the molybdenum content of the pastures is high, licks are provided containing 1 to 2 per cent. $CuSo_4$ (Cunningham, 1955).

It is important that the existence of copper deficiency should be confirmed by blood and also, if possible, by liver analysis before copper is given either therapeutically or as a prophylactic. Indeed, there is no justification for copper therapy unless this has been done and there is a very real risk of toxicity if therapy is applied without confirming the existence of a deficiency.

Response to Treatment

The results obtained in Experiments 4 and 5 show that deficient cattle make a dramatic response to treatment, as assessed in terms of improvement in clinical condition, weight gain, and blood copper levels. Experiment 5 (Fig. 11) shows, however, that copper therapy alone will not achieve this. Spectacular improvement will not occur unless the animals also have an adequate food supply of high nutritive value. Thus cattle treated with copper in October, when the pastures were still good both quantitatively and qualitatively, responded by showing an average daily weight gain of 3 lb. during the next 44 days, whereas a similar group of cattle treated in December, by which time both the quantity and quality of the herbage had deteriorated, made an average daily weight gain of only 1 lb. during the 42 days following treatment, although symptoms were alleviated.

The Mineral Content of Pastures

The results of pasture analyses add little to existing knowledge other than the occurrence of a seasonal variation in the levels of copper and molybdenum respectively, a finding which does not appear to have been reported previously in Great Britain. The figures confirm that the pastures apparently contain enough copper to supply the needs of the grazing animal. This supports the generally accepted theory that the deficiency of copper in the animal must be induced by some other factor in the herbage which interferes with copper assimilation. Molybdenum is known to act in this way but the balance of evidence is against it being the causal factor in the fens.

Economic Importance of the Disease

A primary object of these investigations was to attempt an evaluation of the economic effects of copper deficiency in cattle. It is a question of balancing the cost of prevention or treatment against the difference in value resulting from variations in rate of maturing in deficient and normal cattle respectively. One illustration will suffice to show the effect in terms of agricultural output. The figures given in Table VII show that deficient cattle at 30 months of age might be expected to average 936 lb. live-weight, as against 1,277 lb. for normal animals of the same type, a difference of roughly 3 cwt. In effect, this means that by preventing copper deficiency the cattle are worth about £20 per head more at 2½ years of age. What does it cost to achieve this? Assuming the method of prevention chosen to be that of feeding copper as anti-teart cake during the winter, in order to afford complete protection it would be necessary to feed the pregnant cow so that the calf starts life with a normal copper status. The calf would also require cake during its first and second winters. The rate of feeding required is ½ lb. per head per day, possibly from November to March inclusive. The total amount required would be 150 days × ½ lb. daily × 3 winters, giving a total of 225 lb., or roughly 2 cwt., at a cost around £4, showing a direct profit of £16 per beast. But this, of course, is only part of the story. Beasts of 936 lb. at 30 months of age would need to be kept on for at least another year with the additional labour and food costs which this would entail. Further, these calculations take no account of deaths, which occur not infrequently among deficient cattle, particularly in calves. The figures, without being weighted by factors such as these, are still sufficient to make it quite clear that the prevention of copper deficiency in cattle can mean the difference between a profitable live-stock venture and one running at a continual loss.

Summary

An account is given of experiments carried out over a number of years in East Anglia on the prevention and treatment of copper deficiency in cattle. The dosing of pregnant cows with copper was effective in preventing deficiency in the subsequent calf crop. The disease was also prevented in calves by spraying pastures with a solution of copper sulphate. Observations on store cattle from the time they were 12 to 30 months of age showed a striking difference in weight gain between deficient cattle and cattle in which copper levels were maintained around normal. In other experiments, dramatic recovery occurred in deficient cattle following either the intravenous injection of copper sulphate or the feeding of anti-teart cake. Pastures showed a seasonal variation in mineral content, with an inverse relationship between the copper and molybdenum contents, but the copper content was at all times sufficient to meet the needs of the grazing stock, thus supporting the view that copper deficiency in the animal is induced by some factor in the herbage which interferes with copper assimilation.

Acknowledgments.—The writer is indebted to Sir Thomas Dalling, formerly Chief Veterinary Officer, and to Dr. H. H. Green, formerly head of the Biochemistry Department at the Central Veterinary Laboratory, Weybridge, for their encouragement during the early stages of this work, and to Dr. A. W. Stableforth, Director of Veterinary Laboratory Services, for his interest and co-operation throughout, particularly in providing personnel to assist in the trials. Assistance in carrying out the field work was

given at various times by the following colleagues: Messrs P. D. Thomson, D. Buntain, I. D. B. Newsam, and Dr. K. C. Sellers. The haematological examinations were carried out by Mr. R. F. Sellers and the analytical work by the staff of the biochemistry department of the central laboratory at Weybridge. Special thanks are due to Dr. R. Allcroft and Miss G. Lewis of that department. The photographs used for the preparation of Figs. 9 and 10 were taken by Dr. Allcroft during a joint visit to see deficient cattle on the Norfolk fens before treatment was commenced.

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The President's Address to Congress.—*Concluded.*

body whose members may treat animals—whether they belong to the rich or the poor—was reaffirmed at the highest level.

It is necessary, in view of that wider audience to which I referred earlier on, to make clear the essential importance of diagnosis. I do not have to tell an audience of veterinary surgeons that without a correct diagnosis proper treatment is hardly feasible. And diagnosis does not rest upon conscientiousness or goodness of intention, but upon the years of scientific training which only we, within the animal world, undergo. In any case, as you all know, our profession has an honourable tradition, going back over the years, of helping impecunious animal owners, either by reducing our normal fees or even in appropriate cases by remitting them altogether.

We have always been very willing to co-operate with any animal welfare society which would provide conditions within which we could accept service, and our long relationship with the R.S.P.C.A. and with other welfare societies is ample evidence that this has been achieved. There is no reason at all why we should not reach a useful working relationship with the P.D.S.A. if that organisation desires it, and we have shown ourselves ready at any time to enter into discussions which will lead to that end.

Special Conferences

In recent years it has become our custom to hold conferences on special subjects of particular interest, and it was with much satisfaction that I presided last November over a conference dealing with the important problem of Supplements and Additives to Animal Feeding-stuffs, to which a large number of agriculturists and representatives of the animal feeding-stuffs trade were invited. The Conference, for the organisation of which we have to thank the Technical Development Committee, was, it was generally agreed, a most successful one, and I think it did a great deal of good. The publication of its Proceedings as a special issue of THE VETERINARY RECORD made available, in a compact form, a large body of most useful information both for ourselves and for those interested in livestock farming and ancillary undertakings.

Reference was made by one of my predecessors during the time of his presidency to the purchase of what he described as our present dignified home at Mansfield Street, when he stated that it was our intention to add to the dignity of our new council chamber by the purchase of suitable furniture. That promise has been amply fulfilled, thanks to the generosity of the constituent divisions of the Association, and we now have a Headquarters of which we can justly be proud. I am quite sure that Mr. A. M. Graham and his colleagues at the time, who were responsible for the acquisition of these premises, must now regard them with the greatest satisfaction; we of the British Veterinary Association are particularly appreciative of the very excellent work they did.

There are, of course, many other subjects to which I might usefully have referred to-day, but in the rather limited time at my disposal I have dealt with those which I deemed were of topical interest. I must, however, just add this. We, as a profession, are rather prone to hide our light under a bushel, and to keep our fellow men in a state of quite astonishing ignorance of what we do and how we do it. People do not realise how the work of veterinary surgeons touches their everyday life at innumerable points—the quality and quantity of the food they eat, the woollen clothes they wear, the leather shoes on their feet, their protection from infections of animal origin, and a host of other things.

May I conclude this Address by expressing my deepest gratitude to all of you for the support and loyalty extended to me during one of the most eventful years of my life. Although ever mindful of my own shortcomings, it has, I can assure you, been a particularly happy, if somewhat strenuous year, and it has been a very great privilege indeed to serve as your President. Thank you also for the very patient hearing you have just given me."

The New President of the British Veterinary Association

AT the annual general meeting of the Association, held in Cambridge on August 26th, Mr. J. McConnachie Ingram was unanimously, and with acclaim, elected President of the B.V.A. for 1957-8. He thus enters upon his fifth successive year as an Officer, having been honorary secretary from 1953-6, and junior vice-president from 1956-7. The honour now accorded him will give wide satisfaction, and is the fitting and proper outcome of his experience in the Association's affairs, his great knowledge of, and contribution to, the profession, and his personal prestige and popularity.

The office of President becomes, every year, a more exacting one. The rapid growth of the Association, its increasing commitments, and the constantly widening sphere of its influence call for arduous labour at the highest level, while the number of official engagements requiring the President's personal attendance have become, of themselves, a heavy call both on time and effort. Thus, for the principal of a large practice to find—or rather to make—time for B.V.A. business over several years, and then to undertake the most responsible office of all is something for which every member must be grateful. In these days the Presidency is not undertaken without considerable self-sacrifice and even pecuniary loss.

Though resident in the South of England for a number of years, Mr. Ingram is a Scotsman like several notable Presidents of recent years. He was born in 1905 in Huntly, Aberdeenshire, of a farming family, and had practical experience in agriculture before beginning his veterinary studies. Commencing his education at the Gordon School, Huntly, he passed, by scholarship, into the North of Scotland College of Agriculture, and thence to the Glasgow veterinary school where he graduated in 1928. His early experience in practice was first with Mr. Jas. Anderson of Keith, Banffshire, next with Professor D. Imrie, whose pupil he had been, and finally with Mr. A. H. Andrew for whom he managed a practice in Hertford from 1931 to 1936. Two years later he became a principal, taking over from Mr. Arnold Spicer, F.R.C.V.S., the practice in Oxted which bears his own name.

Mr. Ingram's rise in the profession has been no easy one. He has had none of the advantages of a ready-made family practice, of affluence or of influential support. He has built up an enviable practice, and an enviable reputation as a clinician, entirely by his own efforts. He has been referred to as "a prince among practitioners," and certainly his colleagues in the field can be sure that this year's President is a practical veterinarian through and through. Apart from the variety of his own experience, he has a particular knowledge of the problems of private practice from his comparatively recent presidency of the Society of Practising Veterinary Surgeons. In addition he is a past president of the South-eastern Veterinary Association, a Fellow of the Central Veterinary Society, and a present member of the Sussex Division of the B.V.A.

He has been a member of the Council of the Association since 1947, having served on a number of Standing Committees including the Technical Development Committee. During the past four years he has regularly attended the Editorial Committee, which has benefited greatly from his practical knowledge, and eminent good sense. He further helped THE RECORD by consenting to be a member of the sub-committee who recommended names for the first awards of the William Hunting prizes for the best clinical papers published during 1956.

Biographical articles usually make some reference to hobbies and outside interests. Beyond a day's racing occasionally—his son has ridden successfully point-to-point—Mr. Ingram's only real interest is his profession. The writer had the privilege of spending a day with him a few months ago, when the amount of work done, administrative as well as clinical, the distance travelled, the speed and assurance of diagnosis and of treatment compelled astonishment and admiration alike. It is no wonder he is *persona grata* with owners of stock and domestic pets over so wide an area. From his early years of training, and by natural inclination, he is at heart an agriculturist, and the language of farming comes to him as familiarly as that of veterinary science.

For one whose natural bent is work in the field Mr. Ingram has, none the less, effective contributions to make in the Council chamber. He is impatient of generalities and examines both problems and projects with conscientious and searching attention. His now long experience as an Officer has given to him a comprehensive grasp of the Association's policies, together with a first-hand knowledge of its contacts and exchanges with other bodies from Parliament downwards, so that his preparation for the Presidential responsibility has been unusually thorough.

Mr. Ingram married in 1935. He has one son, in his first year at the Glasgow School, and a younger daughter. Mrs. Ingram had veterinary contacts among her own relatives through an uncle and a cousin, and her practical knowledge and flair, together with a gracious serenity familiar to a wide circle of friends, have contributed beyond measure to her husband's success in his profession, and will contribute in the coming year to a still wider sphere of activity.

Everyone will wish Mr. Ingram well in the heavy task he has taken up at the unanimous request of his colleagues. Certainly they may feel assured that the new leader of their Association is as typical a veterinary surgeon as could well be imagined, and one whom they may count upon to maintain, at its highest level, the dignity and reputation of their calling.

Mr. J. M. INGRAM, M.R.C.V.S., the new President of the British Veterinary Association.

THE JUNIOR VICE-PRESIDENT OF THE B.V.A.

Members of the Association will learn with pleasure that Mr. H. F. Hebeler has been unanimously elected Junior Vice-President and President-elect. Mr. Hebeler will bring to his office wide experience of negotiations on the Association's behalf, for he was Hon. Secretary of the B.V.A. from 1950-3, and Chairman of the Organising Committee from 1950-7.

He was born in Edinburgh in 1917, and was educated at the Edinburgh Academy, St. Edward's School, Oxford, and the Royal (Dick) School of Veterinary Studies. He qualified in 1940, and at once went into his present practice in Langport.

He has been a prominent member of the Western Counties Veterinary Association since 1941, being elected one of their Council representatives in 1947, and President from 1955-6. He became a member of the Milk Sub-committee of the Somerset C.A.E.C. on its inception in 1949, and has also been a member of the Poultry Sub-committee and the County Committee of the Animal Health Trust.

The Association are indeed fortunate to have as an Officer a veterinary surgeon of such great ability and energy, and the profession will look forward to his term of office with satisfaction.

THE HONORARY TREASURER OF THE B.V.A.

AT the Annual General Meeting last Monday it was announced that Mr. H. E. Bywater, who has been Honorary Treasurer to the Association since 1938, had decided to lay down his office. In point of fact Mr. Bywater had first expressed the hope that a successor might be found for him a year or two ago but he had been persuaded to continue until now. Before handing over to his successor, Mr. E. Cotchin, Mr. Bywater reviewed at some length the financial history of the B.V.A. He said:—

"It may, perhaps, be of some service to our members if I take this opportunity of reviewing the financial progress of the Association since its foundation until the present time. In doing so, it may be as well to remind our members that although the National Veterinary Medical Association—as we were formerly designated—was formed as recently as 1921, we are now holding our 75th Annual General Meeting and Congress. This apparent anomaly is accounted for by the fact that the Association was developed by the union of various local veterinary societies throughout the country whose members had acquired the laudable habit of foregathering each year to discuss matters of mutual interest. In 1910, Professor George Wooldridge undertook the duties of Hon. Treasurer of the Committee which sponsored these annual conclaves and, when in 1921 the present confraternity was established, he continued until 1925 to safeguard our financial—and other—interests. He was succeeded by Mr. Alfred West—a man of shrewd business acumen—who served for 4 years, and from whom Mr. W. G. Wragg took over the office until 1938, when his duties with the Ministry of Agriculture became of such a nature that he was no longer able to be an Officer of ours.

I must pay tribute to my predecessors for the way in which they laid foundations upon which the financial stability of the B.V.A. has been built. Undoubtedly much of the success we enjoy to-day was brought about, and fostered by, the acquisition in 1921 of the proprietorship of THE VETERINARY RECORD. The only assets the N.V.M.A. possessed in those days were THE RECORD and the enthusiasm of our pioneer members, though this was tempered by a cautious estimate of the market value of the newly acquired publication.

Whilst not detracting from the yeoman service rendered by our first Hon. Treasurer in connexion with our purely financial transactions, I must digress to pay tribute to his outstanding contributions to the whole work of our profession, and to this Association. We rejoice that he is still with us. Professor Wooldridge must have had many difficulties with which to contend in those early days, but once they were overcome our monetary problems began to be solved, and a steady improvement continued until shortly before I took office.

I must also record the debt owed to many other members of the profession who were actively associated with the early days of our foundation. Their names are too numerous to mention, and I would hesitate to select individuals in case I might inadvertently—or through lack of knowledge—fail to give recognition to all who so richly deserve praise. These pioneers gave not only their time and energy, but also from their pockets. They received no travelling or other pocket expenses in those early days. It was truly a labour of love.

We must also recognise the service rendered by the late Mr. Geoffrey Livesey. He has never received the credit due to him for organising, in 1932, the affairs of the Association whilst he filled, for so short a period, the office of General Secretary. He it was who reorganised our office routine, such as it then was, and laid the foundations for the growth of our present methods of dealing with day-to-day financial business.

Early Growth of the Association

The initial financial birth pains and teething troubles were, fortunately, not unduly prolonged, and within a relatively short time our accounts were satisfactorily balanced and a small balance was carefully harvested year by year. Although Alfred West was only in office for 4 years he brought with him that shrewdness which proved so markedly successful in his own private affairs. It was he who stressed the fact that the function of the Hon. Treasurer was to build up a suitable reserve as an assurance against adverse times, and in pursuit of that objective he insisted upon every economy which was not obviously prejudicial to advancement. It was not until the early 1930's that signs became evident that expenditure was creeping up and was cancelling out income—due, I fear, to a lessening of appreciation by some of our leaders of the necessity of economy. My immediate predecessor, in presenting the annual accounts during his last year of office, had to report an adverse balance of £270, and when I was confronted with the accounts which were produced shortly after I assumed office, I found we had a deficit of nearly £400. Obviously something had to be done, and at

my suggestion an ' economy sub-committee ' was formed with Mr. Arnold Spicer as chairman and Sir (then Mr.) Thomas Dalling as secretary. This sub-committee thoroughly scrutinised the financial position and, following their report, measures were adopted which resulted in the necessary economies. Thus, at that juncture, the need to increase membership subscription rates was averted. The work—indeed the existence—of ' this economy sub-committee ' has almost completely been forgotten, but we should always remember the work and the valuable services its members rendered to the Association at a critical time.

Although, when I assumed office, expenditure exceeded income, basically we were financially stable. Owing to the careful husbanding of our resources at the beginning we had by no means a negligible reserve of capital invested. The Surplus Account, as we now term it, amounted to nearly £15,000. In my early days as your treasurer, we had an income of just over £7,500 a year. Of this, some £4,200 was derived from slightly more than 2,100 members paying the basic annual subscription of 2 guineas. Sales of THE VETERINARY RECORD, priced at 1s. per copy, to approximately 500 institutions and non-members, yielded a little under £1,800.

The War Years

With the advent of war in 1939, the activities of the Association in many, but by no means all directions, quietened down. We ' ticked over ' for a while in so far as our routine activities were concerned. We had, however, not been inactive in considering the probable consequences of warfare. When the newly formed National Air Raid Precautions Committee for animals started to function in earnest, it was to this Association that they turned for office accommodation and clerical assistance. The reduction in the amount of routine Association work enabled our staff to help considerably; and this continued even when we were deprived of the services of Mr. Fredericks, and later a junior member of his section of the staff, upon enlistment in the armed forces.

The general reduction in our own office work coupled with the reduction in size of THE VETERINARY RECORD through paper rationing, resulted in an overall reduction in expenditure; for although prices had commenced to rise, the inflationary trend was only in its infancy. The nett result was, that during the war years we had a steady surplus of income over expenditure which enabled us to improve our reserves. This, incidentally, was in spite of the birth of the ' Survey Committee ' and the expenses associated with the ' Survey Scheme,' which was so enthusiastically fostered and developed during the Presidency of the late Harry Steele-Bodger. Our investment had initially been depleted by a grant of £1,000 to the Victoria Veterinary Benevolent Fund to be used to help practitioners who might suffer in consequence of enemy action. Fortunately this sum was more than ample to meet all contingencies, and eventually no less than £800 of it was refunded. We were also at that time underwriting the activities of N.A.R.P.A.C.

Experiences during the war period emphasised to members of the profession the necessity for a strong and virile body to foster and preserve their interests, and by 1944 our membership had risen to over 2,800, and subscriptions (still based upon the original 2 guineas) reached £5,400. Sales of THE VETERINARY RECORD remained fairly constant but following an increase in price to non-members to 1s. 6d. as against 1s. per copy the total income had risen to nearly £2,000. Increased advertisement rates yielded £3,400. In that year N.A.R.P.A.C. discharged its financial obligations to us, and we were thus able to make a donation of no less than £2,500 to the newly-launched Veterinary Educational Trust—or as it is now styled, the Animal Health Trust—as well as to find £1,200 successfully to oppose in the House of Commons, a Bill, promoted by one of the animal welfare societies, which had passed through the House of Lords despite opposition by the Royal College of Veterinary Surgeons.

Our surplus account still remained at much the same level as in pre-war years, but our investments showed a slight improvement at £17,000.

With the advent of peace the activities of the Association continued to expand with even greater momentum; but our increased activities were accompanied by the general rise of prices. It became obvious that, if we were to continue to expand and to improve, we must acquire additional revenue, and the membership subscription was accordingly raised in 1947 to the present standard rate of 4 guineas.

I must confess that, having received more income, I was not agreeable to see it dissipated unprofitably, and I was never quite as keen as some of my colleagues to embark on schemes of expenditure which the possibly more progressive leaders considered desirable. Rising costs, especially in connexion with THE VETERINARY RECORD which had expanded its size with the easing of paper control, began again to create problems and it soon became apparent that we must find a solution. We therefore substantially increased our journal's advertising rates, and contrary to some predictions this went hand in hand with a greater quantity of business. Instead of THE VETERINARY RECORD threatening to become a liability, it developed into a valuable asset. Indeed, a large part of our surplus during the past few years has been earned by THE RECORD, and it is imperative that this prosperity should continue if our subscription rates are to remain reasonable.

The surpluses thus harvested were of very material assistance to us in the purchase and adaption of 7, Mansfield Street, which we were enabled to complete without disturbing our invested capital.

Post-war Developments

In the post-war years, we have seen our membership increase to 4,400 with a subscription income of over £16,300—nearly 4 times as much as in 1938. The circulation of THE RECORD to non-members has increased to over 1,400 copies per week which, allied to the increase in price to 2s. per copy, yields over £6,700 or 5½ times as much as in 1938. Our advertisement revenue is now over £17,000 or 8½ times that of 1938. Our total income of £43,426 is more than 500 per cent. greater than that of 1938.

At the same time, of course, there has been an

enormous rise in expenditure. Whereas the printing and despatch of THE VETERINARY RECORD cost something over £3,600 in 1938, the cost in 1956 was 5 times greater at £18,830. Such expenses as we paid to Officers and special committees amounted to a mere £600 in 1938, but to-day similar expenses cost us £3,700 a year. The cost of running our annual Congress in 1938 was £153, in 1955 it had risen to £1,200. The last 2 Congresses were in the region of £2,400 each. Very obviously we must look into this item of expenditure which costs us about £6 for every member of the B.V.A. who graces our Congress by his presence. We also now supply our members with free copies of the handbooks which are produced by the labours of the Technical Development Committee and which are, I believe, a most useful source of reference to the veterinary profession. This service runs away with a few hundred each year—but it is probably one of the best returns for money spent that the Association has. Outgoings in all other directions have shown comparable increases over the years.

We have spent over £40,000 in investments (the market value of which is now only £31,000). However, we have invested not to speculate, but to secure an income; and market fluctuations do not affect interest yields in stock already held and as a reserve against eventualities. It is in this latter aspect that market values may affect our position, should the necessity arise to realise some hard cash—which is an eventuality we must take care to avoid.

We now have our own house, the lease of which is shown in the accounts at the very conservative book value of £15,000. In addition we have also found from revenue over a few short years, some £12,000 to meet the cost of acquiring, repairing, and adapting the building to our special needs. We are comforted by the knowledge that the net rents from our tenants will, eventually, enable us to recover the last named sum. Our surplus account now stands at £45,390, nearly 3 times that of 1938.

I am very conscious of the fact that the pound sterling has no longer the purchasing power of the pre-war pound, and that the immediate saleable value of our Government holdings are considerably depreciated; but the Association is financially more stable than it has ever been and, in any case, we now possess permanent headquarters of which we can justly be proud and which should prove a valuable long-term investment, increasing in value with inflationary tendencies, in contradistinction to our Government investments.

Conclusion

In conclusion, I would like to express my thanks to all our staff, without whose loyal devotion to duty no such progress could have been possible, and since I am dealing with the financial aspect of our affairs, I would especially mention Mr. Fredericks, who has been longest in our employment, and upon whom falls the main responsibility for keeping our books. By encouraging him to adopt modern aids to his routine office work, we have been able to ensure that the work of recording all the multifarious transactions involved in the larger membership, with the varying rates of subscriptions, the increased sales, advertising revenue, and general expenditure has been smoothly performed without any material increase in the staff of the ' general office.'

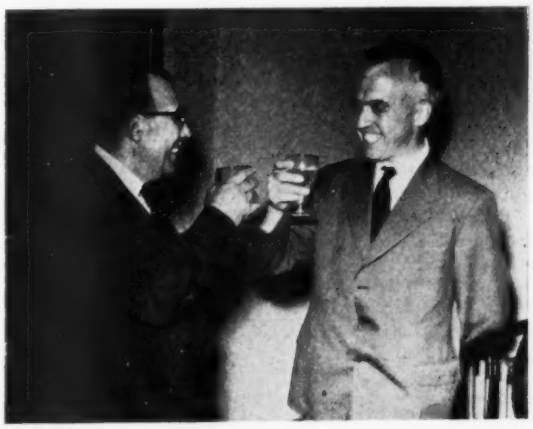
My successor will assume office with the knowledge that we are financially more secure than ever before. Mr. Cotchin, however, will need to be ever vigilant, and must have the support and assistance of the other Officers and of the Council, in maintaining a constant restraint in expenditure if progress is to be maintained and past achievements secured and consolidated. It is not easy to achieve financial stability from ' scratch,' but all too easy to dissipate savings. Wise spending is a necessity, but only in so far as the resources available permit.

Mr. President, ladies and gentlemen, I thank you for your patient hearing; I hope I have not bored you. I wish the British Veterinary Association long life and increasing prosperity and, to my successor, a successful and meritorious term of office. I know he is fully capable of carrying on the good work if only you will give him a measure of that support which has always been given to me."

Following Mr. Bywater's resignation, Mr. E. Cotchin, M.R.C.V.S., College Reader in Veterinary Pathology at the Royal Veterinary College, was unanimously elected Hon. Treasurer.

Nineteen Years' Service

Mr. Bywater has rendered outstanding service to the B.V.A. during the past 19 years. It is to his prudent policies, financial acumen, and resolute judgment, as much as to any other individual factor,



The retiring Hon. Treasurer, Mr. H. E. Bywater, toasting his successor, Mr. E. Cotchin.

The President's tribute to Mr. Bywater, which formed an item of the Annual General Meeting, will be published, with full details of the meeting, next week.

that the Association owes its present position. When he took office the membership, the sales and advertisement of this journal, and other sources of revenue were but a fraction of what they are to-day; nor could it have been envisaged that, by the end of his term, we should enjoy as our headquarters the amenities

of a gracious house in one of the most desirable parts of London, the purchase and equipment of which had not disturbed our invested funds. Each time he enters it, Mr. Bywater may reflect with justifiable pride upon his own great contribution towards its acquisition.

The post of honorary treasurer can never be other than exacting. It is always easier—and more pleasant—to say yes than to say no; but it is the latter which most frequently falls to a treasurer's lot. There may have been times when colleagues were disappointed, or standing committees felt themselves in some degree thwarted, by Mr. Bywater's iron grip on the purse strings. Yet the soundness of his judgment is written again and again in the history of the past 19 years. He has served his colleagues well, and in addition to a tangible mark on their esteem which he is to receive in the near future, his elevation to honorary membership of the B.V.A. is his very just reward, together with the knowledge of a task fulfilled.

Mr. E. Cotchin

He is succeeded in office by Mr. E. Cotchin, Reader in Pathology at the Royal Veterinary College. Mr. Cotchin was educated at Bedford Modern School, whence he passed, by scholarship, to the College, qualifying in 1940. He has served on the Council of the B.V.A. for the past 5 years, being a member of the Technical Development Committee and of the Editorial Committee, whose chairmanship he now relinquishes on being elected an Officer. He is a past treasurer of the Association of Veterinary Teachers and Research Workers, has been treasurer of the College Students' Union for the past 5 years, and is President for 1957-8 of the Central Veterinary Society.

News and Comment

THE OPENING OF CONGRESS

THE proceedings were opened by The Rev. George Willis, B.V.SC., M.R.C.V.S., who invoked the Divine blessing on the Congress. The President then invited the Mayor of Cambridge, Councillor White, to extend a welcome to delegates on behalf of the City.

The Mayor said it was a great honour and pleasure to extend a civic welcome to delegates attending the 75th Annual Congress of the British Veterinary Association at Cambridge, the seat of so much learning. He had read that the veterinary art was of great antiquity, and he had noticed that veterinary surgeons were known as " doctors of asses and oxen " as long ago as 2300 B.C. He had also learnt that in the 14th century veterinary practitioners were known as Marshals and Master Marshals of London at a trade guild organised in the year 1356.

His Worship extended a particular welcome to the ladies who, he felt sure, would find a great deal of interest in Cambridge with its ancient buildings, wonderful colleges, and open spaces. " We hope you will have an enjoyable and profitable time while you are here," he concluded, " and will take happy memories away with you. We wish you well."

Lord De La Warr's Address

The President next invited the Earl De La Warr, P.C., G.B.E., D.L., to perform the official opening ceremony. In so doing Lord De La Warr first thanked the Association for the honour which had been accorded him in being invited to open the Congress. He also congratulated the President on the way in which the Congress had been organised.

The British Veterinary Association's Congress, he said, was not a meeting of a trade union. It was a meeting of members of a great profession with great traditions, which played a notable part in our national life. Not only was it an honour to be asked to address the Congress, but it afforded an opportunity of meeting a great many old friends. It was some long time since he and the President last met. It was in a small hotel on the Yorkshire moors when the President brought along a group of Yorkshire and Lancashire farmers to try to tell the Ministry what it was necessary to do about hill sheep farming. That was 10 or 11 years ago, but before and since then it was not necessary to recall the great record of service to the profession which Mr. Callender had given. Members must all be very proud of having a most worthy President. (Applause.)

" I do not think that I can say I am meeting Mr. Ingram *again*," continued Lord De La Warr, " because we are always meeting. I always groan when I see him on the farm because I know something has gone wrong! Many is the animal he has saved for me, and many the tip he has given me in connexion with his other more private activities! I must say he gave me a bit of a surprise the other day when he came in to see a cow with a twisted gut. He looked at the animal, sent for ropes and so forth, then turned her over on her back and proceeded to indulge in 5 minutes' rock and roll with her! The next morning I do not know who was more surprised to find that the cow was perfectly all right again."

Lord De La Warr recalled that in the past he had had an opportunity of meeting the profession under many different colours. There were the years at the Ministry of Agriculture, the very many happy and proud years working with the Agricultural Research Council, and then in his own farming activities, both in this country and in Rhodesia. In that connexion, he said he sometimes wished that more British veterinarians were able and willing to go and carry the colours overseas. " It is often a matter of great regret to some of us to see those concerned in Rhodesia having to go to other sources in order to fill up the ranks, although you certainly have sent some very

worthy representatives. I am one of those who believes very strongly in maintaining British standards and traditions all over the world, and of course especially in our Empire and Commonwealth, and I do not believe there is a better ambassador whom you can send than an absolutely first-class member of a great profession such as yours."

Thanks from the Farming Community

Another reason why he felt particularly happy at having been asked to open the Congress was that it afforded him, as a member of the farming community, an opportunity of saying a brief though sincere "thank you" for what the profession had given to the agricultural industry. Everyone knew the fantastic changes which had taken place in the farming industry even since the war, and it must be a very proud thought in the minds of all members of the veterinary profession just how great was the contribution which their profession had made to this progress in farming. When considering a few of the instances where great progress had been made, it might be said that possibly the greatest step forward had been the manner in which the curse of tubercle had been dealt with. There were over 70 per cent. of attested herds at the present time, and a number more were on the way. There was also that old enemy, mastitis. One could also recall that which had been accomplished by the veterinary profession in connexion with pulpy kidney in sheep, lamb dysentery, and many other diseases. At the same time one thought of what the veterinary profession could do for pigs with swine fever if farmers would only make more use of the vaccine. It was possible to go on in such a strain *ad infinitum*, therefore it was only natural that anybody coming from the farming industry should want to say "thank you."

It would also be appropriate to offer congratulations, for he had seen enough of the veterinary profession over the last few years to know of the very great increase in status which the present generation of veterinarians had built up in the country. The profession was becoming more and more scientific as was almost everything else. That applied to every profession and industry. Probably by virtue of the greater fund of knowledge which had to be acquired and investigated, the veterinary profession was being driven more and more into a considerable degree of specialisation. Of course, in so far as that made it possible to have more thorough investigation of certain problems, it must be all to the good; but as a layman he wondered whether he might offer a word of caution. It was necessary to bear in mind that life was not a series of specialised separate processes. The animal was not a mass of separate organs and functions. The mystery of life was in co-ordination and in the relationship between the different organs and functions, and it was necessary to have a good all-round man to deal with them.

Unity with the Animal Health Division

One thing which he had noticed particularly, as a layman, was the close working together of the Ministry of Agriculture, the Animal Health Services—research side—and those working in the field. That was obviously of immense importance and must add greatly to the richness of the profession and what the profession was able to give to the farmer and to the public.

He had no desire to touch on a difficult subject, but it had come to his notice that some members of the profession felt that they were losing a great deal of possible knowledge by being denied access to material in slaughter-houses. He had been in politics too long to want to go into the matter very deeply, but one could not help feeling that there was a great deal of material there which might be allowed to members of the veterinary profession in order that they might extend their field of knowledge. The subject was no doubt receiving careful consideration by the authorities, and he felt sure that consideration should continue to be given and a decision arrived at.

Lord De La Warr concluded by congratulating the retiring President, Mr. Callender, on a great year's work as President, and by wishing the very best of good fortune to Mr. Ingram, the incoming President. (Applause.)

WELCOME TO DELEGATES AND GUESTS

The President said he desired to extend a cordial welcome to all delegates and guests, and particularly to those from overseas who included: Lieut.-Col. Lasher from the American Veterinary Medical Association; Mr. D. N. Thornton, Australian Veterinary Association; Dr. P. J. G. Plummer, Canadian Veterinary Medical Association; Dr. K. Seidel, Danish Veterinary Association; Dr. M. Sterne, South African Veterinary Medical Association; Mr. C. V. Dayus, New Zealand Veterinary Association; Major C. R. Chadwick, Iraq Veterinary Association; Mr. W. G. Beaton, Kenya Veterinary Association; and Mr. W. R. Nunn, Uganda Veterinary Association.

A full list of delegates, as read at the meeting, will be published next week.

The President, Mr. E. R. Callender, in a relaxed moment with Mr. G. N. Gould. President of the R.C.V.S.

THE NUTRITION SOCIETY

The Scottish Group of the Nutrition Society is holding a symposium on " The Nutrition of the Very Young " in Dundee on Saturday, September 21st, 1957. The papers to be read deal with both infants and young farm animals. Those interested, who are not already members of the society, are invited to write for further particulars to Dr. J. Davidson, The Rowett Research Institute, Bucksburn, Aberdeenshire.

OPENING OF THE TRADE EXHIBITION

The President, in opening the trade exhibition, cordially welcomed the representatives of the many firms who were exhibiting at Cambridge. The exhibition assumed greater importance every year, and there was no doubt it had become a most essential part of Congress, offering as it did an excellent opportunity of seeing at first hand the latest developments in connexion with drugs, instruments, books, and a host of other things of vital importance to veterinary surgeons. The Association was grateful to the exhibitors for their excellent display, and members would welcome the opportunity during the course of the week of meeting them in friendly discussion.

Mr. G. N. Henderson, speaking on behalf of the exhibitors' Committee, thanked the President for his kind remarks, and expressed the hope that those delegates who visited the exhibition would be interested in what they saw. The current exhibition was probably the largest ever to have been held at Congress, which might well be an indication of the increasing interest in the veterinary profession being shown by the pharmaceutical industry—an interest which it was hoped would go some way towards improving the products available and which, in turn, would lead to an improvement in the treatment of disease and its control.

On behalf of the Exhibitors it gave him great pleasure to ask Mrs. Callender to accept a small gift. (Applause.)

The President, having thanked Mr. Henderson, then moved, with the Earl and Countess De La Warr and the remainder of the platform party, into the trade exhibition, where Mrs. G. N. Henderson presented a bouquet to Mrs. Callender. Thereafter the stands were visited, the party spending an appreciable time at each.

THE COMMERCIAL EXHIBITION

The details of the following stand were very reluctantly held over until this week owing to pressure on space.

Burroughs Wellcome & Co. (The Wellcome Foundation Ltd.)

Emphasis this year was placed on the control of canine distemper and hard pad disease, a facet of veterinary medicine wherein this Company has gained international recognition.

The exhibit paid tribute to the original work of Laidlaw and Dunkin and to the Field Distemper Council, which led to the large-scale production at The Wellcome Research Laboratories, of Distemper Prophylactics Methods I and II. These prophylactics served the veterinary profession well until they were superseded in 1953 by " Epivax," the non-virulent egg-adapted distemper vaccine.

To-day, as 30 years ago, The Wellcome Research Laboratories are the only laboratories in Britain producing an agent for active immunisation against distemper.

In addition to illustrating various stages in the production of " Epivax," the B.W. & Co. exhibit had as its tailpiece an enlarged photograph of the 1957 Cruft's Supreme Champion, Volkrijk of Vorden, who was immunised with " Epivax " as a puppy.

Amongst the literature distributed at the Congress, pride of place went to the B.W. & Co. folder. As in previous years, this had been specially designed for the Congress and bore local colour and features; it incorporated a guide to Cambridge and described the veterinary contribution of Dr. John Caius, a graduate and benefactor of the University. The folder containing a selection of literature has been sent to all members of the Veterinary Profession, with a cordial invitation to visit the B.W. & Co. Stand, No. 5.

Willows Francis Ltd.

The focal points of the display were the Intramammary Antibiotic Soxamycin and the renowned local anaesthetic Xylotox veterinary, together with Venagmin the established veterinary blood coagulant.

Three new products were introduced to the many friends who visited the stand, i.e., Neovagon a veterinary uterine bolus for retained placenta and uterine hygiene. Neosulphentrin an improved product for the control of intestinal bacterial infection in all species, and Vioxel for the control of infection and pain in the disease of the ear and eye, in small and large animals.

Among many items that aroused interest were (Willows long-acting ACTH), Xydermo for the treatment of dermatosis of undetermined origin in small animals, the new formulated Euthesate for the humane destruction of animals by intravenous or intraperitoneal injection.

Literature and information were also available on other well-known Willows veterinary products including Rumoxane the modern remedy for bloat. Kebovel for the prevention and treatment of ketosis and samples were available of the new plastic pack of Willows Teat Plugs.

Finally a comprehensive range of veterinary instruments was displayed including the new Xylotox veterinary cartridge syringe.

PERSONAL

Births

JONES.—On August 21st, 1957, at the Garrett Anderson Maternity Home, Hampstead, to Shirley, wife of Bruce V. Jones, M.R.C.V.S., a son, Julian Patrick.

SUTHERLAND.—On August 20th, to Jill, wife of D. J. Bruce Sutherland, M.R.C.V.S., of 81, Benton Street, Hadleigh, Suffolk, a daughter.

Marriage

ROULSTON—LYON.—On August 8th, 1957, at Kings' College Chapel, Old Aberdeen, Thomas L. Roulston, M.A., ED.B., 3, Moat Street, Londonderry, to Edith Lyon, M.R.C.V.S., 4, Queen's Gardens, Aberdeen.

WHITELEY—EMMETT.—On August 14th, 1957, at St. Aiden's Church, South Shields, Co. Durham, Hedley Whiteley, B.V.SC., M.R.C.V.S., of Halifax, Yorkshire, to Ann Elizabeth Emmett of South Shields, Co. Durham; future address, 5, Westwood Road, Beverley, E. Yorkshire.

THE REGISTER OF VETERINARY SURGEONS

Under the provisions of Section 6 of the Veterinary Surgeons Act, 1881, and Section 13 of the Veterinary Surgeons Act, 1948, the name of the under-mentioned was removed from the Register on August 21st, 1957:—

Bhattacharjee, James, 4, Poyaza Street, Kanbe P.O., Insein District, Burma.